BROILER ASCITES SYNDROME: A REVIEW

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Summary

A summary of the most recent theories on pathogenesis of the ascites syndrome in broiler chickens is presented. Methods used to experimentally reproduce the syndrome are compared, with caution expressed on extrapolation of results from studies using hypobaric conditions to field situations where the major inducer of the syndrome is hypothermia. The role of genetic selection in the increase in incidence is discussed and indications drawn from local work indicating that differences in susceptibility between broiler breeds may be most related to selection for greater muscle mass relative to cardio-pulmonary capacity. Recent research results are discussed.

I. INTRODUCTION

The ascites syndrome or pulmonary hypertension syndrome of broilers has been the subject of an enormous amount of research over the past six to eight years, reflecting the importance that this condition has achieved in worldwide chicken meat production. An international seminar on this disease was held in Arkansas in January 1997 allowing the most up to date progress to be presented. The multifactorial nature of the ascites syndrome is readily evident just from studying the program for this meeting, covering possible interactions of genetics, hatchery management, growout management, environment, nutrition, metabolism, growth rate and other diseases.

In this paper just a few aspects of the syndrome will be reviewed, with particular emphasis on how they relate to the occurrence of the disease in Australia and recent research into its control.

II. CURRENT THEORY ON PATHOGENESIS

Wideman and Bottje (1993) have reviewed this area comprehensively. Genetic selection for improved growth rate, feed efficiency and greater meat yield has resulted in a proportion of birds within a broiler population having an inherent potential to outgrow their cardio-pulmonary capacity. With an increase in the relative size of a rapidly growing muscle mass to organ ratio, the bird requires additional blood to be supplied by the heart to support this growth. This increased blood flow leads to an increased blood flow through the lungs (pulmonary hypertension). This increased pulmonary blood flow leads to capillary transit time reduction and the bird becomes more susceptible to an imbalance of blood perfusion and respiratory diffusion, resulting in hypoxaemia.

Hypoxaemia leads to a polycythemia as a result of erythropoietin stimulation. This increases blood viscosity, leading to the need for greater cardiac effort and a further increase in pulmonary hypertension. Progression of the inherent distension and hypertrophy of the right ventricle causes right atrio-ventricular valve insufficiency and congestive heart failure, ascites and oedema is the end result.

Wideman and Bottje (1993) further point out that this progression can be affected by two commonly encountered environmental problems. Firstly, low barometric pressure due

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to altitude can result in hypoxia. In response to hypoxia, the pulmonary arterioles undergo constriction. This can substantially increase pulmonary vascular resistance and enhance pulmonary hypertension. The other potent ascites inducer is hypothermia. This stimulates increased metabolic rate and, thus, heat production. Systemic pre-capillary arterioles dilate to allow increased tissue oxygen uptake. This response requires increased cardiac output which in turn elevates pulmonary blood pressure.

III. EXPERIMENTAL REPRODUCTION OF ASCITES SYNDROME

To evaluate hypotheses on factors involved in the ascites syndrome, it is necessary to work with an experimental system that will consistently reproduce the disease. The syndrome first became apparent in countries growing chickens at high altitudes (>1500m) [eg. Mexico, South America, South Africa, Kenya] and remained as a problem only in these areas for some years.

Appropriately, many researchers adopted low barometric pressure, either by using high altitude or simulated high altitude in hypobaric chambers. Several workers (Owen et al., 1990; Witzel et al., 1990) developed successful models based on this method of ascites induction. Ascites levels can be exceptionally high under these conditions, especially if concurrently affected by low environmental temperatures.

Over the last 10-12 years, however, ascites has become a common problem in birds reared at lower altitudes, including Australia. The condition appears identical and often results under the hypobaric model are assumed to give identical performance under low altitude conditions. This may not always be a safe assumption, as will be alluded to below.

Ascites may be induced at low altitudes using ambient temperatures below the chicken's thermoneutral zone. Julian et al. (1989) produced significant increases in ascites incidence by lowering ambient temperature to 13°C after 22 days of age. In a factorial experiment Verstegen et al. (1989) induced ascites by gradually decreasing ambient temperature from 30°C down to 16°C by 14 days and further to 11°C by 30 days. A comparable group kept above 25°C till 14 days and then subjected to a decline to 11°C at 30 days were less affected indicating that low ambient temperatures in the first two weeks predispose to later ascites. Field observations support this finding (Groves, 1991).

Deficiencies in ventilation and, hence, questions of air quality have also been incriminated as being involved in the production of the ascites syndrome. Maxwell et al. (1989) described a higher incidence of cartilaginous and osseous nodules in the lungs of birds in poorly ventilated chicken houses. Lung damage has been incriminated in the aetiology of ascites and ascitic chickens have a higher number of these nodules in their lungs, but a causal association has not been demonstrated.

Ventilation deficiencies and the build up of toxic gases have been proposed as causes of ascites but this has come more from inference than from controlled experiments. Dale and Villacres (1986) noted that increased ventilation had been one of the few effective methods to reduce ascites in high altitude situations as long as cold stress was avoided. Closer to sea level, however, ventilation has been less successfully employed to control the syndrome. Julian and Wilson (1992) measured the oxygen and carbon dioxide concentrations in air in numerous pens of broilers and were unable to show any differences in gas concentrations in pens with high or low levels of ascites. In this study, oxygen concentration inside the house was only marginally reduced compared to the outside air (20.50% compared with 20.85% respectively) even though carbon dioxide levels were elevated compared to outside air (around 0.37% compared to 0.04% respectively).
Shlosberg et al. (1992) compared a well ventilated and an extremely poorly ventilated shed at low altitude (690m). Poor ventilation, which resulted in ammonia concentrations of 70 ppm and high humidity, was only able to decrease oxygen concentrations to 20.4% (compared with 20.7% in the well ventilated control and 20.9% outside) and no differences in ascites incidence due to ventilation differences were found.

Jones (1995) studied different oxygen concentrations without altering barometric pressure and concluded that sub-optimal oxygen concentrations did not increase ascites susceptibility.

Maxwell (1990) reported that reduced ventilation in winter coupled with increased gas-fired heating could lead to carbon monoxide concentrations of up to 70 ppm which would lead to hypoxia and hence ascites. He also suggested increased carbon dioxide, ammonia, humidity and dust could be involved.

Balog et al. (1994) induced ascites using an extremely low ventilation rate (beginning at 0.003m³/minute/bird at 5 weeks) and was able to somewhat reduce the disease under these conditions using ceiling fans, concluding that the fan prevented air stratification and, thus, diluted toxic gases.

These last two examples would have to be considered extreme for commercial conditions. Experience in Australia concurs with Israeli results (Shlosberg et al., 1992) that ventilation plays little part in ascites and cold temperatures are the major inducer.

Ascites in Australia appears to be largely a New South Wales phenomenon with occasional problems in southern Queensland. It would be expected that the colder states would be most afflicted by ascites. Generally, shedding in South Australia and Victoria is designed to handle cooler temperatures, having a larger proportion of controlled environment sheds and making more use of hot air brooding (space heating) methods. In NSW sheds are predominantly open sided relying on natural ventilation and using radiant heaters (spot brooding) for early warmth. Queensland enjoys much milder winters and hence this type of housing copes much better. Increased ventilation under Queensland conditions often incurs only a small degree of cold stress.

Thus, it would appear that improvements in ventilation may only assist where low barometric pressure is the major inducing factor for ascites, which makes some sense as oxygen partial pressure is already reduced at altitude and further respiratory stress exacerbates the hypoxia. However, where the main factor causing ascites is low temperature (i.e. the hypoxaemia is caused by a non-respiratory demand for oxygen) where the effects on oxygen concentration will be negligible, improvements in air quality offer no decrease in the incidence of ascites.

IV. EFFECTS OF GENOTYPE ON ASCITES SUSCEPTIBILITY

Since the earliest emergence of the ascites syndrome it was obvious that certain strains and crosses of commercial broilers were more susceptible to the condition (Hargis and Odom, 1990; Groves 1991). It has been suggested that with intense genetic selection for improved growth rate, feed efficiency and meat yield the relative size and performance capabilities of the broiler's cardio-respiratory system has been diminished, possibly to a point where it has reached its metabolic limit.

Several studies have been undertaken to compare chickens of various types and ascites susceptibility. Vidyadaran et al. (1987) and Vidyadaran et al. (1990) compared anatomical respiratory parameters of a modern egg layer strain with the red jungle fowl, the putative progenitor of the domestic chicken. These workers demonstrated that the modern layer has a 33% smaller lung volume/body weight ratio and its blood gas tissue barrier is
28% thicker than its wild ancestor. Julian (1989) measured the lung volume of a modern broiler strain and demonstrated a decline in the lung volume/body weight ratio with age. Actual figures from these three studies have been adapted for comparison in Table 1.

Although different measurement techniques were used by these researchers, it can be judged that the lung volume/bodyweight ratios for modern layers and broilers are similar and both are reduced compared to the red jungle fowl. This observation gives credence to the theory described above on genetic selection for performance traits selecting against lung size. This is interesting as genetic selection of layer and broiler strains have taken quite different paths for many years, targeting totally different performance traits. Lung size per se cannot be the full answer however, as the lung volume/bodyweight ratios of broilers appears to be similar to that of layers which are not susceptible to ascites under normal chicken rearing conditions.

<table>
<thead>
<tr>
<th>Bird Type</th>
<th>Age</th>
<th>Lung Volume / Bodyweight (mm³/g ± SEM)</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Red Jungle Fowl</td>
<td>7 d</td>
<td>25.81 ± 2.80</td>
<td>Vidyadaran et al.</td>
</tr>
<tr>
<td></td>
<td>30 d</td>
<td>17.18 ± 2.69</td>
<td>1987</td>
</tr>
<tr>
<td>Commercial Layer</td>
<td>7 d</td>
<td>10.59 ± 0.93</td>
<td></td>
</tr>
<tr>
<td></td>
<td>30 d</td>
<td>15.81 ± 0.57</td>
<td></td>
</tr>
<tr>
<td>Red Jungle Fowl</td>
<td>Adult</td>
<td>18.10 ± 1.90</td>
<td>Vidyadaran et al.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1990</td>
</tr>
<tr>
<td>Commercial Layer</td>
<td>Adult</td>
<td>14.65 ± 3.17</td>
<td></td>
</tr>
<tr>
<td>Broiler</td>
<td>7 d</td>
<td>19.9* ± 1.76</td>
<td>Julian, 1989</td>
</tr>
<tr>
<td></td>
<td>24 d</td>
<td>17.4 ± 0.87</td>
<td></td>
</tr>
</tbody>
</table>

* Converted to mm³/g.

A trial comparing five broiler lines available in Australia (Groves and Cross, Unpublished results) showed no significant difference in lung volume/bodyweight in normal birds at 28 days (Table 2).

<table>
<thead>
<tr>
<th>Broiler Strain</th>
<th>Ascites Susceptibility</th>
<th>Lung Volume / Bodyweight (mm³/g ± SEM)</th>
<th>Right Ventricular / Total Ventricular Weight Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>J</td>
<td>High</td>
<td>9.64 ± 0.36</td>
<td>0.229 ± 0.023</td>
</tr>
<tr>
<td>K</td>
<td>High</td>
<td>9.24 ± 0.18</td>
<td>0.214 ± 0.017</td>
</tr>
<tr>
<td>L</td>
<td>High</td>
<td>10.55 ± 0.96</td>
<td>0.231 ± 0.010</td>
</tr>
<tr>
<td>M</td>
<td>Low</td>
<td>10.79 ± 0.86</td>
<td>0.208 ± 0.013</td>
</tr>
<tr>
<td>N</td>
<td>Intermediate</td>
<td>12.25 ± 0.89</td>
<td>0.215 ± 0.015</td>
</tr>
</tbody>
</table>
Huchzermeyer et al. (1988) compared four broiler strains at high altitude and found that the two more ascites-susceptible strains had higher ventricular/total ventricular weights than the two lines of lower ascites susceptibility. As can be seen from Table 2, the study on broiler strains in Australia failed to show any significant differences in this parameter.

Martinez et al. (1992) in a little known study compared heart valve diameters of two broiler strains with a layer strain (White Leghorn) at various ages. At 3 weeks of age both atrio-ventricular valves and the aortic semilunar valve of the broilers were larger than those of the layer strain; however, the pulmonary semilunar valves of the broilers had smaller diameters than those of the layers. This area needs more research as this lack of development of the pulmonary semilunar valve could be consistent with the development of right sided congestive heart failure (pulmonic stenosis).

Other anatomical measurements were taken on the five broiler strains studied in Australia (Groves and Cross, unpublished results; Table 3).

Table 3: Anatomic and carcass characteristics of broiler strains in Australia.

<table>
<thead>
<tr>
<th>Strain</th>
<th>Keel Length/wt 28 days</th>
<th>Shank Length/wt 28 days</th>
<th>Breast Fillet, % carcass 49 days</th>
<th>Thigh Fillet, % carcass 49 days</th>
<th>Drumsticks, % carcass 49 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>J</td>
<td>62.5&lt;sup&gt;a&lt;/sup&gt;</td>
<td>35.4&lt;sup&gt;a&lt;/sup&gt;</td>
<td>21.3&lt;sup&gt;b&lt;/sup&gt;</td>
<td>12.6&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>14.52&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>K</td>
<td>66.4&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>28.9&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>21.7&lt;sup&gt;b&lt;/sup&gt;</td>
<td>13.4&lt;sup&gt;b&lt;/sup&gt;</td>
<td>13.4&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>L</td>
<td>69.4&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>39.2&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>20.5&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>12.8&lt;sup&gt;a&lt;/sup&gt;</td>
<td>14.3&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>M</td>
<td>71.9&lt;sup&gt;b&lt;/sup&gt;</td>
<td>41.3&lt;sup&gt;b&lt;/sup&gt;</td>
<td>19.7&lt;sup&gt;a&lt;/sup&gt;</td>
<td>11.9&lt;sup&gt;a&lt;/sup&gt;</td>
<td>16.2&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>N</td>
<td>69.2&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>39.5&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>21.3&lt;sup&gt;b&lt;/sup&gt;</td>
<td>12.0&lt;sup&gt;a&lt;/sup&gt;</td>
<td>14.2&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<sup>a,b</sup> - Means with different superscripts differ significantly (P<0.05).

The strain with the lowest ascites susceptibility (M) tended to have a larger keel and shank, and lower meat yield than the highly susceptible breeds (J, K and L). This indicates that differences in ascites susceptibility between these broiler strains has more to do with a higher muscle mass to bodyweight ratio than with variations in lung or heart capacity at this genotypic level.

Metabolic differences have also been studied in broiler lines of varying ascites susceptibility. The scientific literature reveals a number of studies comparing broiler strains which have been selected for faster growth or superior feed efficiency and leanness in relation to their thyroid function in response to lower ambient temperature. Scheele et al. (1991) and Scheele et al. (1992) provided evidence that a line selected for improved feed conversion was relatively hypo-thyroid at low temperatures compared with a line selected only for growth rate and that this line had an inability to increase its feed intake in response to cold. Decuytere et al. (1992) furthered the link with ascites and thyroid function and suggested that supplementing birds with thyroxine could be a method of identifying susceptible lines.

Jones (1994) working with similarly selected but different lines of birds suggested that it may be the ability of the efficient feed conversion line to maintain its growth at low temperatures without a corresponding increase in feed intake which predisposes this line to ascites.
V. RECENT STUDIES ON ASCITES CONTROL

There have been a number of reports of improvement in the incidence of ascites by the use of medications, either in feed or water. The target of much of this work has been to improve pulmonary blood perfusion and/or oxygen diffusion capacity in the lungs. Success here would obviously benefit birds with the potential for compromised cardio-respiratory capacity.

Owen et al. (1994) showed that by altering the acid-base balance of the feed ascites incidence under hypobaric conditions could be altered. Acidification of feed, by adding ammonium chloride, caused a slight increase in ascites while alkalinization, by adding sodium bicarbonate, significantly reduced ascites under these conditions. The mechanism of action was suggested as being a possible metabolic alkalaemia causing variable pulmonary vasoilation and a consequent decrease in pulmonary arterial pressure. A metabolic acidosis would cause the reverse response.

Wideman et al. (1995a) used furosemide in drinking water under cold temperature conditions and reduced mortality due to ascites. Furosemide is commonly used in mammals with congestive heart failure. It is a potent diuretic that leads to reduced pulmonary arterial pressure and also acts as a pulmonary vasodilator. The copious diuresis would present practical difficulties for the commercial use of this drug, not to mention regulatory difficulties with its use.

Wideman et al. (1995b) supplemented broiler feed with 1% L-arginine and were able to reduce ascites losses. L-arginine in excess over dietary requirements is required as a substrate for nitric oxide production, the latter being a potent endogenous pulmonary vasodilator, thus decreasing pulmonary arterial pressure. This is a natural product but its cost would be somewhat prohibitive for general use at present.

These reports highlight the role of reducing pulmonary arterial pressure as a means of controlling the condition.

Vanhooser et al. (1995) used metaproterenol, a bronchodilator, in drinking water under varying oxygen concentrations and temperatures. At lower temperatures this drug reduced ascites significantly.

Therapeutic manipulation of bronchiolar and pulmonary capillary characteristics can successfully modify the incidence of the ascites syndrome, confirming the role of cardio-pulmonary compromise in the condition. Whether these findings can be employed commercially is yet to be seen. In the meantime, while genetic progress in selection against susceptibility to the syndrome is awaited management practices which can minimize the effects, such as temperature control, lighting programs and dietary measures can be implemented (Groves, 1996).

REFERENCES


