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Review

Nutritional and managemental control of ascites syndrome in poultry

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Ascites or pulmonary hypertension is a multifactorial syndrome, caused by interactions among genetic, environmental, dietary and physiological factors. Forced selection to achieve faster growing chickens has improved genetic potential; but, due to some anatomical and physiological limitations, the same improved potential could have adverse effects on bird health. Impaired oxygen supply to sustain a continuous fast growth rate may increase the risk for a higher incidence of ascites syndrome. The impaired oxygen supply will stimulate the development of many compensatory mechanisms in cardiopulmonary systems, which, in turn, bring ascites syndrome. Management practices to provide thermoneutral environment, limit growth rate (such as: feed restriction, nutrient density and diet form), use of antioxidants and omega three fatty acids normalize red blood cell structure and function to match the demand of oxygen requirement, which in turn can reduce the chance of ascites.

Key words: Broiler chicken, ascites syndrome, nutrition, genetic, management.

INTRODUCTION

The term "ascites" actually refers to the fluid accumulation in abdominal cavity. Ascites (pulmonary hypertension syndrome, or water belly) is a metabolic disorder, characterized by hypoxaemia, increased workload of the cardiopulmonary system, central venous congestion (Luger et al., 2003), an excessive accumulation of fluid in body coelomic cavities (Olkowski et al., 1999), hypertrophy of the right ventricle and a flaccid heart (Riddell, 1991), and finally death (Luger et al., 2003). Although the incidence of this metabolic disorder in well-managed flocks is very low, it causes important economic losses to the poultry industry (Maxwell and Robertson, 1997; Balog, 2003; Bin et al., 2007).

Ascites syndrome, first reported in flocks of broiler chickens reared at high altitudes in Bolivia (Hall and Machicao, 1968), may result from one or more of four physiological changes that cause an increased production and/or decreased removal of peritoneal lymph (Balog, 2003). Obstruction of lymph drainage, decreased plasma oncotic pressure, fluid leakage secondary to increased vascular permeability, and, increased portal pressure secondary to right ventricular failure or liver damage may all result in ascites (Julian, 2005). Since plasma proteins, especially albumin, are mainly responsible for blood oncotic pressure, some researchers (Wise and Evans, 1975; Bowes et al., 1989) explained decreased plasma proteins in ascites-sensitive broilers or those with ascites. Decreased plasma protein could be a result of loss of high-protein lymph from the liver or a stop-eating process due to right ventricular hypertrophy. Vascular damage and subsequent leakage of fluid and proteins through the vascular epithelium can be caused by viral and/or bacterial infections, chemical toxins, chlorinated hydrocarbons and some phenolic compounds, coal-tar derivatives, dioxin and pentacholorophenol (Balog, 2003). Increased vascular hydraulic pressure could be a result of hepatic pathologies, right atrio-ventricular valve pathologies, pulmonary hypertension and miscellaneous cardiac pathologies. The avian respiratory system possesses rigid lungs of fixed volume (Fedde, 1984), which do not expand or contract with each breath as mammalian lungs do. The blood and air capillaries form a network that allows the small

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blood capillaries of the lung to dilate only very little to accommodate increased blood flow (Julian, 1998). On the other hand, an increase in contractility of the myocardium, due to an elevated preload, increases pulmonary vascular resistance. Pulmonary vascular resistance increases after load leads to pressure overload and ventricular hypertrophy. This also distorts atrio-venticlar valves and produce regurgitation. As a result of volume overload of the ventricle and the resultant haemodymanimic pressure, ascites develop (Currie, 1999).

Ascites symptoms in broiler chickens include generalized oedema, fluid accumulation in the pericardium, hydropericardium (pericardial effusion) (Olkowski et al., 2003) in the abdominal cavity (Balog et al., 2003), epicardial fibrosis, lung oedema, enlarged, flaccid heart (Balog et al., 2003), hypertrophy and dilation of the heart, especially the right ventricle (Decuypere et al., 2000), variable liver changes, hypoxaemia, pale comb and higher blood haematocrit (Luger et al., 2003). These symptoms indicate that a large number of organs (including the heart, lung, liver, etc.) are involved in the disease.

Ascites is a multi-faceted syndrome caused by interactions between physiological (for example, O_2 demand), environmental (for example, altitude) and management (for example, ventilation and disease status) factors (Baghbanzadeh and Decuypere, 2008). Genetic, physiological, environmental, and management factors all seem responsible for ascites syndrome.

GENETICS AND ASCITES

Poultry with faster growth rates are more likely to develop ascites. Genetically, the modern broiler, especially male broilers, seems to be more prone to develop ascites. Total mortality due to ascites has been reported higher in the male parent lines (Dewil et al., 1996). This is probably due to extreme selection for either the growth rate or the feed conversion ratio, which puts high demands on the metabolic processes and on the oxygen demand (Decuypere et al., 2000), although oxygen requirement is affected by genetic factors other than growth rate. Birds selected both for low food conversion ratio (FCR) with low rates of heat production that were stimulated to a higher heat production by a low ambient temperature had difficulties in adapting to environmental changes. It has also been shown that the highest incidence of ascites occurs in broilers that combined low FCR with fast growth rate, whereas in broilers with either slower growth or higher FCR, the incidence of ascites was much lower. A low FCR in fast-growing birds was attributed to low values of heat production. Moreover, birds selected for a combination of both fast growth and low FCR had low PO_2 and high PCO_2 in venous blood at low ambient temperature compared with the slower growing birds (Decuypere et al., 2005).

Generally, poultry breeders have been very successful in selecting growth-related traits of broilers over the past few decades. The breeding industry has now taken up new challenges, and efforts are being directed to produce stock adaptable to a wide range of environments and to decrease the incidence of metabolic and physiological disorders, of which ascites syndrome is an example (Pakdel et al., 2004). Birds with high levels of oxygen saturation level in the blood (SaO_2) have a reduced susceptibility to ascites and sudden death syndrome. One breeding industry has incorporated the routine assessment of birds for the SaO_2 into its selection strategy. This is done by removing individuals and families that are found below the average levels of SaO_2. Over time, this has led to a significant increase in the SaO_2 levels of the blood, thus reducing the susceptibility of ascites. There are a few reports about genetic parameters for ascites-related traits. Lubritz et al. (1995) demonstrated favourable heritabilities for fluid accumulation in the abdominal cavity and the ratio of right ventricular weight to the total ventricle weight. They suggested that selection based on these traits, which were measured under cold conditions, would be effective to reduce the incidence of ascites.

GROWTH RATE AND ASCITES

There is a direct correlation between metabolic rate and ascites levels. A fast growth rate increases the demand for oxygen and hence the workload of the heart. The modern chicken has been intensely selected for higher growth rates and so indirectly for a high rate of protein synthesis, which requires more oxygen (Decuypere et al., 2005), increased feed efficeincy (Decuypere et al., 2000; Pakdel et al., 2002), egg production (Decuypere et al., 2000) or meat yield and breast percentage (Hoving-Bolink et al., 2000). Modern strains of broilers are able to achieve market weight in 60% less time than broilers of 40 years ago. Nevertheless, the pulmonary and cardiac capacity of modern broilers is very similar to the old broiler strains, which forces their cardiopulmonary system to work very close to its physiological limit (Lorenzoni et al., 2006). The lung capacity does not always meet the oxygen demands necessary for rapid growth. This results in impaired ability to regulate the energy balance under extreme conditions, such as low ambient temperature or high altitude (Luger et al., 2003). If the lung of the chicken grows less rapidly than the rest of the body, it could result to hypoxia and ascites (Julian, 2000).

Therefore, adapting good management practices is vital for fast growing broilers. Growers who have recurring problems with ascites may find it beneficial to control early growth rates. The first 3 weeks of a bird's life are metabolically stressful as bone and muscle growth are greatest at this time. If growth is reduced during this period, oxygen demand will also be reduced. Birds whose growth is controlled early may have a

stronger cardiovascular system going into the finisher phase. However, any restriction of early growth should be exercised with caution. Achieving adequate growth during the first 7 days is vital and so any growth control should be implemented after 7 days of age. Starter diets should remain unchanged to ensure that the day-old chick has the best possible start. Effective control of growth rate after 7 days of age can be achieved by reducing nutrient intake either by reducing the nutrient density of the diet or by changing feed form from a pellet to a mash. Any feed program must be managed properly and should only be considered once optimal management is assured. It is also important to consider that feed control may result in an overall reduction in growth rate. Any management strategy aimed at reducing early growth is therefore only likely to be economically viable when it is properly managed and where the occurrence of ascites is severe.

NUTRITION AND ASCITES

The broiler growth rate has been found to have a direct relationship with susceptibility to ascites (Camacho et al., 2004, Ipek and Sahan, 2006). Manipulation of the diet composition and/or feed allocation system can have a major effect on the incidence of ascites. In most instances, such changes to the feeding programme influence ascites via their effect on growth rate. Major nutritional factors, including high nutrient density rations, high feed intake and feed form are known to influence the occurrence of ascites in broilers (Balog et al., 2000; Coello et al., 2000; Bolukbasi et al., 2004; Ozkan et al., 2006).

Although the growth rate can influence metabolic diseases during the entire period of broiler production, research has shown that the early period is particularly important (Camacho et al., 2004). A reduced growth rate from 3 to 14 days of age not only benefits bird health during that period, but also later when the growth rate is as fast or faster than birds that have not experienced slower early growth. These suggest that an early day of production represents an important developmental period in poultry meat stocks. Recently, management strategies have been investigated to alter the growth curve of meat stocks with the objective of reducing the incidence of metabolic diseases while maintaining competitive production traits. The methods included quantitative and qualitative feed restriction, altered feed form and environmental management.

Feed restriction

Feed restriction has proven successful in reducing ascites, but the degree of restriction required to control health problems needs to be balanced with the time required to reach market weight and other effects on bird productivity. Feed restriction reduces growth at a critical time in a broiler chick's lifecycle when it is the most susceptible to metabolic disease due to its high oxygen demands (Arce et al., 1992; Balog et al., 2000; Coello et al., 2000; Ozkan et al., 2006, 2010). Camacho et al. (2004) has concluded that feed restriction and microelement supplementation at 7 days of age reduced mortality due to ascites. Despite some of the advantages of feed restriction as a treatment to reduce ascites, adverse secondary effects might result in reduction of the availability of nutrients and pigmentation precursors, which may have a direct effect on weight gain, muscle mass and the profit-cost relationship. These effects could be more pronounced if the restriction programme was not correct (Camacho-Fernandez et al., 2002).

Nutrient density

Reducing the concentration of nutrients in a diet can reduce the growth rate, with the effects most pronounced from 0 to 21 days of age, during the time when birds cannot totally adapt intake to lower feed nutrient content. If diets remained balanced to energy content, the effect of nutrient density on growth rate is relatively small unless the decrease in density is very large. However, even moderately lower nutrient density reduces mortality due to ascites (Camacho-Fernandez et al., 2002).

Diet form

Most meat birds are fed crumbled or pelleted diets to achieve maximum growth and feed efficiency. Feeding mash reduces growth rate (1 to 2 days to market), mortality and condemnations due to metabolic disease. However, this type of programme may not be economically acceptable in all areas and has been demonstrated to increase the incidence of pendulous crops. Broilers that consume pellet feed have frequently been shown to have higher incidences of ascites than broilers that consume the same diet in mash form (Bendheim et al., 1992; Bolukbasi et al., 2005).

Omega-3 fatty acid

It has been found that supplementation with fish oil reduces blood viscosity and right ventricular hypertrophy in rats (Archer et al., 1989). However, other studies (Hulan et al., 1989) reported a reduction in the growth rate of birds fed red fish meal as a source of omega-3 fatty acids. This is important, because the incidence of ascites can be reduced by slowing the growth rate of broilers (Julian, 1993). The increased content of unsaturated fatty acids probably increases the fluidity of the erythrocyte membrane and alters membrane function to increase the deformability of the erythrocytes and potentially help reduce the incidence of ascites. This could explain the reduction in whole blood viscosity under hypobaric conditions with feeding of flax oil. These factors together would decrease the

resistance to blood flow and improve the movement of the erythrocytes through the capillaries, thus improving oxygen transport and decreasing ascites (Walton et al., 1999).

Antioxidants

The elevated production of reactive oxygen in broilers prone to ascites may potentiate the development of the disease or aggravate the disease as it occurs (Enkvetchakul et al., 1993). For chickens, the first line of defense against reactive oxygen is endogenous antioxidants such as tocopherols, glutathione and ascorbic acid. The levels of glutathione and α-tocopherol and γ-tocopherol are decreased in the mitochondria of an ascitic broiler, suggesting reactive oxygen is produced at the primary site of energy transduction (Cawthorn et al., 2001). Ascorbic acid and glutathione concentrations are reduced in both the liver and lung of broilers that have been reared in ascites-promoting conditions, signifying their utilization against reactive oxygen production in these tissues. Therefore, the change in the antioxidant status of the broiler during ascites progression is observed in conjunction with increased markers of reactive oxygen-mediated tissue injury, indicating a state of oxidative stress during ascites.

Researchers have attempted to alleviate the onset of ascites by increasing the antioxidant status of the broiler before exposure to ascites-promoting conditions. Broilers that received a vitamin E implant that released a total of 15 mg α-tocopherol from 0 to 3 weeks of age immediately before exposure to ascites had significantly reduced ascites-induced mortality than placebo-treated broilers (Bottje et al., 1995). Liver and lung concentrations of α-tocopherol in healthy vitamin-E-treated birds were increased, providing the bird with additional protection from reactive oxygen. Healthy vitamin-E-treated birds had plasma lipid peroxide values lower than placebo-treated birds in the same conditions, indicating the enhanced protection that vitamin E provides against lipid peroxidation. The vitamin E implant reduced ascites-induced mortality, probably by providing an enhanced antioxidant defence against the reactive species production that otherwise, causes tissue damage and promotes ascites progression. In contrast to the results obtained with vitamin E implants, supplementing broiler diets with vitamin E did not reduce ascites-induced mortality (Bottje et al., 1997; Villar-Patino et al., 2002). Vitamin C, supplemented in broiler feed at 400 mg/kg feed, reduces lipid peroxidation in cardiac tissue as well, but does not affect ascites-induced mortality.

ENVIRONMENTAL/MANAGEMENT FACTORS AND ASCITES

Altitude

The most obvious environmental factor that play a role in ascites development of broilers is high altitude. A high incidence of ascites can occur when broilers are reared at altitudes high enough to substantially reduce the partial pressure of oxygen (Owen et al., 1990; Wideman et al., 2003). The effect of high altitude is a decrease in the partial pressure of oxygen than that at seal level. Exposure to a lower partial pressure of oxygen will lead to an increased workload on the heart. As such, when birds are exposed to low atmospheric oxygen levels (high altitude), pulmonary blood vessels constrict and pulmonary vascular resistance increases (Wideman, 1997). This immediate increase in pulmonary arterial pressure can cause right ventricular hypertrophy and eventually result in ascites syndrome (Wideman et al., 1998). In this situation, it is critical to ventilate correctly and provide as much oxygen to the flock as possible.

Cold temperature

Strong correlation between cold temperature and ascites has been recognized (Sato et al., 2002; Ipek and Sahan, 2006). Exposure to cold periods that place birds outside their thermo-neutral zones will increase the demand for oxygen as birds are forced to use energy to keep warm. Cold temperatures increase ascites by increasing both metabolic oxygen requirements and pulmonary hypertension (Bendheim et al., 1992; Julian et al., 1989; Stolz et al., 1992). Wideman and Tackett (2000) attributed this increase in pulmonary arterial pressure to a cold-induced increase in cardiac output, as opposed to being caused by hypoxaemic pulmonary vasoconstriction. The effect of the timing of a cold stress on ascites development in broilers indicates that exposure to cold temperatures during brooding has a lasting effect on ascites incidence (Julian, 2000; Groves, 2002). The consensus appears to be that cold stress during the first 2 weeks of life affects the bird's metabolic rate for several weeks and increases their susceptibility to ascites.

Maintaining adequate brooding temperatures are critical to the prevention of Ascites. Correct and monitored temperature during brooding, along with a good minimum ventilation program from placement, will help reduce and in some cases eliminate any ascites problems seen later in the grow-out period.

Lighting

Broilers are usually grown on a near-continuous lighting schedule so that feed consumption and growth rate can be maximized. Early studies in photoperiod manipulation reported a decreased growth rate for broilers raised with a step-down lighting programme (Classen et al., 1991). It was hypothesized that limiting the number of hours of light will slow growth slightly and will reduce activity that requires additional oxygen, and may actually improve feed efficiency (Julian, 1990a, b).

Subsequent studies on the effect of longer dark periods or intermittent lighting indicated that similar to feed restriction, photoperiod manipulations can decrease the incidence of ascites syndrome (Julian, 1990b, 2000; Buys et al., 1998; Hassanzadeh et al., 2000, 2003).

Air quality and ventilation

The most influential environmental factor affecting ascites in broilers is the oxygen content of air brought into the poultry house. Suboptimal ventilation and inadequate litter management in poultry houses lead to low environmental oxygen and higher toxic gases (carbon monoxide, carbon dioxide or ammonia), which may have detrimental effects on the respiratory or cardiovascular systems of birds and promote ascites development (Wideman, 1998). It has also been suggested that the environmental dust will be inhaled by the birds thus leading to irritation and reduced efficiency of the airways. Poor air quality, dust and respiratory diseases all predispose birds to ascites by causing respiratory damage. Ventilation rates must supply enough air to replenish the oxygen consumed and ensure the adequate removal of waste gases. Correct litter management in conjunction with appropriate ventilation helps to maintain air quality.

Incubation

Oxygen requirement is the most critical trigger of ascites in broilers (Julian, 2000). High metabolic demands together with decreased availability of oxygen may lead to hypoxaemia and ascites (Wideman, 2001). Ascites susceptibility is particularly pronounced during the period of rapid juvenile growth when the metabolic rate is very high (Decuypere et al., 2000). Rapid growth increases the oxygen requirement, cardiac output and blood flow, and may result in increased pulmonary arterial pressure primarily by increasing the metabolic demand for oxygen (Julian, 2000; Wideman and Tackett, 2000). Chicken embryos grow rapidly over the last 7 days of incubation, resulting in a 60% increase in the oxygen consumption during the interval between the start of pulmonary breathing and hatching (Decuypere et al., 2000; Sahan et al., 2006). Therefore hypoxia, known to be involved in the occurrence of the ascites syndrome, could arise in the chick embryo during the interval between internal pipping and hatching (Dewil et al., 1996).

Oxygen supplementation from 18 to 21 days of incubation could be used as an effective means of improving hatchability of broiler eggs. Oxygen supplementation during incubation could also increase the embryonic growth rate and 1-day-old chick weight (Sahan et al., 2006). Rouwet et al. (2002) demonstrated that chronic hypoxia during embryonic development induces structural and functional cardiovascular abnormalities (for example, left ventricular dysfunction) in the near-term chick embryos. These abnormalities may be responsible for the increased mortality of embryos incubated under high altitude. Different degrees of ventilation during incubation may therefore interact with genotype and egg shell characteristics (which determine gaseous exchange) to affect the total incubation time and, thereby, influence the susceptibility to ascites and related physiological responses in later postnatal life (Chineme et al., 1995).

pH

Ascites is ultimately caused by an imbalance between the oxygen supply to the body tissues and the oxygen requirement of the tissues (Julian, 1993). In mammals, acidosis causes vasoconstriction, while alkalosis causes vasodilation, which affects pulmonary arterial pressure and pulmonary hypertension. The blood pH also affects the affinity of haemoglobin for oxygen in the lung and release of oxygen to the tissues (the Bohr effect). A decrease in blood pH lowers the oxygen affinity of haemoglobin, which encourages release in the tissues, while increased blood pH increases oxygen affinity to increase haemoglobin saturation in the lung (Issacks et al., 1986). The feeding of excess chloride or sulphate has been shown to depress blood pH and bicarbonate levels in chickens (Ruiz-Lopez and Austic, 1993), while feeding bicarbonate would be expected to increase blood pH. It has been suggested that broiler chickens that have a high metabolic rate may be in a state of metabolic acidosis when they are on full feed (Julian, 1993). Several workers have shown that fast-growing birds have lower blood oxygen concentration than slow-growing birds; likewise, birds on full feed have lower blood oxygen than food-deprived birds (Fedde et al., 1998; Julian and Mirsalimi, 1992; Reeves et al., 1991).

Feeding low chloride/high bicarbonate diets results in a decrease in pulmonary hypertension. Conversely, feeding diets with high chloride content tends to increase the incidence of ascites. Increased blood pH would increase oxygen haemoglobin affinity, which is low in fully fed broilers, probably because of metabolic acidosis. It has been demonstrated that decreased blood pH results in increased pulmonary arterial pressure in mammals and this may also be true in birds. Increased blood pH can improve the loading of oxygen by haemoglobin in the lung due to the Bohr effect. It therefore appears that supplementing broilers with bicarbonate may be beneficial in fast-growing birds with very high oxygen requirements and high production of carbon dioxide, as long as the decreased pH normally present in the muscles that facilitate oxygen unloading is not affected (Squires and Julian, 2001).

SUMMARY AND CONCLUSION

Ascites is a multifactorial syndrome, caused by

interactions among environmental, physiological and genetic factors. Nutritional and management practices to limit growth rate, such as: feed restriction, nutrient density, diet form, use of antioxidants and omega three fatty acids, can reduce the chance of ascites.

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