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Ascites in Broiler: Updates

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ABSTRACT

In recent years, intensive selection has resulted in the development of metabolic diseases, including ascites, in chickens as a consequence of their genetic potential to grow rapidly. Many factors interact to cause ascites, including management practices, environmental conditions, and genetic makeup. In the modern broiler industry, ascites syndrome (AS), also called pulmonary hypertension syndrome (PHS), is regarded as the main reason for morbidity and mortality. Current meat chicken lines have a faster metabolic process, which requires a greater amount of oxygen, particularly when they are fed high nutrient density diets or when they are kept in cold environments, Due to their underdeveloped cardiorespiratory systems, broilers cannot get enough oxygen to meet their requirements and suffer from hypoxemia, which in turn leads to pulmonary hypertension syndrome. A wide range of management practices as controlled feeding protocols, and limited lighting periods, besides adding antioxidants to feed, are outlined for reducing ascites incidence in broiler chickens. Also, the use of higher levels of dietary vitamins such as C and E, besides selenium, is believed to help lower the incidence of ascites. The improvement of low-temperature environmental shelter conditions appears to reduce the prevalence of PHS. Molecular genetics employed to diagnose and select against susceptible broilers' grandparents is being employed to eliminate involved genes.

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INTRODUCTION

Ascites is a disease diagnosed through fluid buildup inside the coelomic cavity, pulmonary hypertension syndrome (PHS) is the more scientific name for the disease. In most cases, ascites is diagnosed between 4 and 5 weeks of age **(Sangeeta and Pubaleem, 2019)**. The males of meat lines are more subjected to PHS than the females of the same lines because of their faster growth and better feed conversion ratio **(Dewil, *et al.*, 1996).** PHS appears to be caused by a combination of factors, including genetic make-up, low environmental temperature, and management practices **(Namakparvar, *et al.*, 2014).** Ascites is a defect in metabolism, regarded as a remarkable reason for financial loss in poultry production due to increased mortalities of 5–7%, mostly of heavy birds at marketing age **(De Smit, *et al.*, 2005)**.

If broiler flocks were given free choice feed with high calorie content, a large number of them would develop AS **(Hadad, *et al.*, 2006; Druyan, *et al.*, 2007b)**. By selecting against the genes of AS susceptibility, the reduction in growth rate that is caused by management would no longer be necessary **Wideman and French (1999, 2000)** got a good result by working on elite and grandparent broiler lines. Both sexes that did not get ascites after PHS-promoting surgery for pulmonary artery blockage were kept and used in the production of resistant progeny. Lately, successful monitoring revealed that mortality in ascetic broilers happened due to a crack in the right atrium **(Olkowski *et al.*, 2007).**

Genetically, the novel commercial broiler chickens are suffering from this illness. Breeding for fast growth and higher feed utilisation results in a meat chicken with a high metabolic rate, but its internal organs such as the heart and lungs struggle to produce enough oxygen to sustain life (Havenstein *et al.*, 1994). The purpose of this review is to (1) describe the pathophysiological changes that occur during ascites. (2) Outline the causes and intervention practices to avoid ascites. (3) Discuss the molecular genetics of ascites.

Pathogenesis

Ascites causes and risk factors

Although the illness has been the subject of extensive research for a long time, the direct cause of PHS remains unknown **(Crespo and Shivaprasad, 2003).** According to the literature, the main causes of ascites in modern broiler chickens are an excess of resistance to pulmonary blood inflow as well as an increased need for oxygen due to the fast growth rate (**Wideman, *et al.*, 2013; Dey, *et al.*, 2018).** Ascites has a highly complex etiology.

Common signs of ascites

Poorly developed birds, water belly or a dilated abdomen, loss of activity, shuttered or dull eyes, sagging wings, and clinically impacted broilers may breathe with their beaks open are common signs of ascites **(Olkowski, *et al.*, 2003).** A possible cyanosis, characterized by skin discoloration, particularly on the head (comb and wattles), muscle cells, variable liver changes, and a higher blood hematocrit may be noticed **(Luger, *et al.*, 2003)**.

Ascites can be caused by a variety of factors, including the following

High altitude

It is the most obvious environmental element that could contribute to the development of ascites in meat chickens. Reduced oxygen partial pressure is a result of high altitude, whether real or simulated. Exposure of birds to high-altitude results in the constriction of pulmonary blood canals and an increase in the impedance of pulmonary blood circulation **(Wideman, 1997**). According to **Wideman, *et al.* (1998)**, a sudden rise in pulmonary arterial pressure has the potential to lead to right ventricular hypertrophy and ultimately ascites syndrome.

Broiler chicken flocks raised at high altitudes were the first to be affected by ascites syndrome **(Hall and Machicao, 1968), which** may be brought on by one or more physiological abnormalities that result in a rise in peritoneal lymph production and/or a reduction in peritoneal lymph clearance. High altitudes caused the mortality of male broilers to reach 30% **(Balog, 2003).**

Low temperature

It is the second-most extensively researched environmental factor in pulmonary hypertension and ascites. Many years ago, there was awareness of the strong association between cold temperatures and heart hypertrophy or ascites. Cold is regarded as a prominent factor in ascites epidemics in backyard flocks **(Kalmar *et al.*, 2013),** due to increased blood flux out of the bird's lungs to provide the body with internal warmth. Ascites is exacerbated by cold weather by raising pulmonary hypertension and metabolic oxygen demands **(Julian, *et al.*, 1989; Stolz, *et al.*, 1992**). Instead of an oxygen deficiency caused by pulmonary vasoconstriction, the cold causes an increase in pulmonary vascular push, which leads to an increase in heart action.On the other hand, high heat leads to increased requirements for oxygen and hence the occurrence of ascites, (**Wideman and Tackett, 2000).**

Microorganisms

Aspergillus fumigatus, a mould that can occasionally be found in the surroundings of all fowl is another harmful agent. This mold's disease, also known as "brooder pneumonia," results in hard nodular regions and mould colonies in the lungs that infect the air sacs and eventually cause ascites to develop **(Dhama, *et al.*, 2013).** Other systemic lesions in the internal organs may overlap with ascites. Salmonella spp. and E. coli are the two most frequent bacteria that cause ascites **(Walid, *et al.*, 2020).**

Nutritional effect on ascites incidence

The occurrence of ascites can be significantly impacted by changing the diet's nutritional makeup and/or the way that feed is distributed. Most often, these modifications to the feeding programme affect growth rate, which in turn affects ascites. Ascites in meat chickens can be caused by significant dietary parameters, such as high feed concentration, and an increase in feed consumption, in addition, to feed structure (shape). Diets low in calories have been suggested as a way to lower the prevalence of PHS **(Coello, *et al.*, 2000; Balog, *et al.*, 2000; Ozkan, *et al.*, 2006).** Feed restriction and nutrient concentration reduction in the diet can limit growth and prevent ascites-related death **(Camacho-Fernandez, *et al.*, 2002).**

The effectiveness of a nutrition regimen can be separated into two categories: the effectiveness of minimizing or preventing ascites and the effectiveness of treating ascites that already exist in a flock (prophylactic effect). Contamination with aflatoxins has a role as a stress factor that helps in ascites incidence, it is found in feed, litter, and liver tissue samples collected from broiler houses that suffer high mortality due to ascites syndrome. (**Westlak and Dutton, 1985).**

Sodium chloride

Ascites is brought on by acute salt intoxication from drinking water. This is due to the fact that it causes a decrease in the ability of erythrocytes to deform which may disrupt blood influx dynamics leading to ascites syndrome ***(Mirsalimi, et al., 1992).***

Antioxidants

Broiler diet with vitamin C supplementation lowers lipid peroxidation in heart tissue in ascetic

birds; vitamin C supplements brought RBC counts to the standard levels **(Ruiz-Feria, 2009)**. The addition of dietary vitamin C brought the RBC counts back to normal formation in ascitic chickens as corticosterone levels continued to rise **(Baghbanzadeh and Decuypere, 2008).** The vitamin E insertion will decrease the likelihood of causing ascites death, most likely by supplying a stronger antioxidant defence against the generation of reactive species that would otherwise cause chronic inflammation and accelerate ascites development. Preserving the health of the heart, blood vessels and other organs requires vitamin E. It is regarded as the primary chain-breaking antioxidant that scavenges oxygen free radicals and prevents further peroxidative cellular membrane damage, according to **Lorenzoni and Ruiz-Feria, (2006).**

Lighting program

According to numerous research studies about the regulating periods of light and darkness in poultry houses, photoperiod alterations can reduce the incidence of ascites syndrome, as happens when feeding is restricted **(Julian, 2000; Hassanzadeh *et al*., 2000).**

Genetics and ascites

Apart from temperature and management factors, broilers' genetic makeup can make them susceptible to PHS due to the moderate to high heritability of the genes linked to the disease. According to research, a limited number of genes have a major role in the heritability of ascites **(Kalmar, *et al.*, 2013).** A prominent role for abnormal innate and adaptive immune responses is shown by ascites in broilers **(Wideman, *et al.*, 2013).** The unique ancestor and undirected mating are responsible for the high level of similarity and inherited disease appearance **(Ezzulddin, *et al.*, *2021*).**

The genetics business has recently encountered modern difficulties, work is being done to develop stock that is capable of adapting to a variety of conditions and reducing the prevalence of mortality problems, and PHS is considered one of these important issues **(Pakdel, *et al.*, 2004).** The heavy new broilers, particularly males, appear to be genetically predisposed to PHS development. The oxygen demand is influenced more by the genetic part than the growth rate, this is likely the result of excessive selection for valuable economic traits such as weight gain and feed efficiency, which place significant requests on the oxygen demand to perform these functions, and genetic variables other than growth rate affect oxygen need **(Decuypere, *et al.*, 2000)**. Birds selected for high feed efficiency and minimum levels of energy production, which were then driven to produce more energy in low environmental temperatures, had trouble adjusting to ambient changes. as well as demonstrated that the occurrence of this syndrome is significantly lower in birds characterized by slow growth and a high feed conversion rate than it is in those with both quick growth and a low FCR**. A. Baghbanzadeh1 and Decuypere, (2008).**The incidence of PHS could be decreased through breeding based on the evolution of these characteristics in cold environments.

Haematocrit and the ratio of the right ventricular weight to the total ventricular weight are two ascites features that could be used as an indirect criterion for reducing the occurrence of broiler ascites **(Wideman, *et al*., 1997**; **Scheele, *et al.*, 2003; Pakdel, *et al.*, 2005).** Selection should have been done, taking into account maternal genetic effects. Before implementing a selection strategy to reduce the incidence of ascites, genetic correlations between production traits measured under commercial circumstances and under cold-stressed circumstances are required.

Genes or markers associated with ascites

In an investigation by **Sufang Cheng, *et al.*, (2021)**, mutant genes were shown to increase pulmonary artery reconstruction and be related to ascites incidence. The investigation turned up the next mutated genes: the ALDH gene family, ACOD1, GGTLA1, IGSF1, DEXH, USP36 (ubiquitin specific peptidase 36), C6orf76, TLT2, SPAG1, CD34, PLEKHA7, and it was discovered the importance of the gene ALDH7A1, which has a major turn on pulmonary blood-vessel restructuring together with vascular constriction, and they all increased vascular resistance, which caused AS to develop. Former investigations have suggested the action of one or two alleles related to arterial constriction has a major role in the development of AS in heavy chicken. **(Lubritz and McPherson, 1994; Anthony and Balog, 2003; Navarro, *et al.*, 2006; Druyan, *et al.*, 2007).**

Krishnamoorthy *et al.*, (2014), mentioned that there are many SNPs spread in several regions on chromosomes 1, 9, 27, and Z that play a role in ascites occurrence. In addition, the region on chromosome 9 between Mega base pairs 12 and 13 had a significant relationship with the incidence of HPS in broilers. An important relationship between the phenotype and the regions (11.9–13.6 and 15.5–16.3 Mbp) on chromosome 9 mostly affects females. Other related genes on this chromosome are AGTR1 (Angiotensin II Type 1 Receptor), UTS2D (Urotensin 2 Domain Containing Protein), and 5HT2B (Serotonin Receptor/Transporter Type 2B) **(Watanabe *et al*., 2006; Djordjevic and Gorlach, 2007; Chung *et al*., 2009).** Further work determined the involved regions on 2, 5, 10, 27, and 28 chromosomes that affect ascites (**Rabie *et al*., 2005).**

Two microsatellite markers used to examine the genotyping in three grandparents of broiler breeder lines detected a linkage with PHS in the Gga 9–13 region, where the gene Angiotensin II Type 1 Receptor exists. **Dey, *et al.*, (2016)** determined a region on chromosome 2 consisting of seventy megabase pairs related to resistance against AS in male broilers has cadherin 6 (CDH6) and melanocortin-4 receptor (MC4R) genes. With significant progress in DNA sequencing, the next generation sequencing is a great pathway for determining all regions and genes responsible for AS in rapidly growing broilers. Employing entire DNA resequencing, **Dey, *et al.*, (2018)** specified single nucleotide polymorphisms in exon 8 and intron 6 of the carboxypeptidase (CPQ) gene on chromosome 2 (about 127 Mbp), related to male resistance. In conclusion, work is still going on to find all the genes related to the disease and seek to get rid of them by selecting the ancestors of commercial broilers.

Ascites management and protection

First and foremost, it's critical to comprehend the fundamental reasons why ascites can occur on a chicken farm. Feed restriction may lessen the impact of the condition in cases of hereditary ascites. Birds with slower growth have lower oxygen requirements, allowing the lungs and heart to deal with the birds' oxygen needs. However, broiler growth performance declines when feed intake is reduced. Feed limitation only has a positive economic impact when ascites incidence is extremely high **(Wideman, *et al.*, 2013; Kamely, *et al.*, 2015).**

Ascites has no effective treatment, and if symptoms appear in birds, death usually happens very rapidly. A diuretic called furosemide can be used to lower ascites mortality. Due to their ability to lower the number of free radicals produced during ascites, selenium, vitamin C, and vitamin E should be used in feeding methods to minimize mortality. **(Vegad, 2004).** Restricted feeding or employing a poor calorie ration over the duration of the breeding cycle are two ways to avoid ascites. As a result, feed limitation inhibits body growth, enabling the heart and lungs to provide the birds with oxygen **(Tottori, *et al.*, 1997).** Rationing will help lower the incidence of ascites. Because pelleted diets have a higher density than mashed diets, broilers can consume more nutrients. To reduce ascites, mash can be fed during the early stage (for 2-3 weeks after hatching) **(Shlosberg, *et al.*, 1991).**

Avoid applying the poultry litter treatment to reduce dust and ammonia **(Terzich, *et al.*, 1998).** They cause serious lung irritation and harm, which reduces the amount of oxygen the bird can obtain from its environment and leads to ascites.

CONCLUSION

The complicated condition known as ascites is brought on by the interplay between physiological, environmental, and managerial factors. By using sound managerial, medical, and nutritional practices, ascites incidence can be decreased. Today, ascites syndrome mostly affects chickens that grow quickly, and it becomes economically significant when it causes a significant amount of mortality (about 5%) at industrial farms that raise broilers. In this investigation, factors relating to humidity, weight, and age appear to combine to produce AS by raising the metabolic rate, which in turn raises PCV. Limiting rapid growth and managing environmental influences is an essential aspect of maximizing poultry farm revenue by decreasing the mortality rate of chickens because of AS. Research has confirmed that weight, age, and sex, in addition to humidity have a close relationship with the spread of ascites disease in poultry fields for meat production. Overall, controlling rapid growth rates through restricted feeding with low feed energy and the use of antioxidants such as vitamins C and E, as well as selenium, in conjunction with a limited lighting programme and environmental management (temperature and ventilation) is the best option for the success of chicken enterprises and avoiding ascites problems.Genetically, the focus should be on eliminating ascites-causing alleles.

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