Simran Singh *et al*, International Journal of Advances in Agricultural Science and Technology,
Vol.5 Issue.6, June- 2018, pg. 9-15 ISSN: 2348-1358
Impact Factor: 6.057
NAAS Rating: 3.77

Ascites Syndrome: A Challenge for Blooming Poultry Industry

Simran Singh[1], Harshit Verma[2*], D. Chakraborty[3]

[1]*PhD Scholar, Department of Animal Genetics and Breeding, SKUAST Jammu- 181102, simranvet@gmail.com*

[2]**Assistant Professor, Department of Veterinary Microbiology, SVPUAT, Meerut- 250110, dr_harshitverma@yahoo.co.in*

[3]*Assistant Professor, Department of Animal Genetics and Breeding, SKUAST Jammu -181102, dibyendu_vet40@yahoo.co.in*

**Corresponding author: dr_harshitverma@yahoo.co.in*

Abstract

Ascites syndrome (AS) or Pulmonary Artery Hypertension, a metabolic disorder frequently perceived in broiler chickens, is primarily the upshot of high pressure in the pulmonary artery. It is marked by augmented pulmonary hypertension, right-ventricle hypertrophy, fluid accumulation in the pericardium and abdominal cavity, increased hematocrit that results from increased red blood cell production (erythropoiesis), and a slump in arterial blood O_2 saturation. In fact 1% of mortality from AS leads to considerable economic losses because it occurs toward the conclusion of the growing period and, thereby affecting heavy birds on which a considerable investment of labor and feed have assimilated. As the high metabolic rate is a key component contributing to the susceptibility of broilers to Ascites. Some of the feasible nutritional strategies such as early age feed or nutrient restriction (qualitative or quantitative) and light restriction lower the growth rate without compromising the final body weight. Optimization of the house temperature and ventilation in cold weather are beneficial management practices to decrease Ascites incidence. Breeding objectives should aim at locating and eradicating all the AS susceptible individuals in the selected population and selecting for high growth rate among the AS resistant ones.

Keywords: Ascites Syndrome, Pulmonary Hypertension, Mortality Nutritional, Management, Breeding

Introduction

Effecting both humans and broiler chickens on a large scale, Ascites syndrome (also known as Pulmonary Arterial Hypertension) remains a clinically challenging disease. It is distinguished phenotypically by increased pulmonary hypertension, right-ventricle hypertrophy, fluid amassment in the pericardium and abdominal cavity, increased hematocrit that results from increased red blood cell production (erythropoiesis), and a decline in arterial blood O_2 saturation (Havenstein *et al.*, 2003 and Druyan *et al.*, 2009). It can be imputed to disequilibrium between cardiac output and the anatomical capacity of the pulmonary vasculature to accommodate ever-increasing rates of blood flow, as well as to an inappropriately elevated tone (degree of constriction) maintained by the pulmonary arterioles. If the pulmonary vasculature is continually distended by fast blood flow to the level that it becomes non-expandable, the circulation hemodynamic blood pressure in pulmonary vessels will elevate inducing pulmonary artery

hypertension. To actuate excessive blood flow throughout the lungs, the right ventricle is administered to higher pressures, further ensuing in right ventricular hypertrophy and failure (Kalmar *et al.*, 2013). Although its occurrence in well-managed flocks is very low, it causes important economic losses to the poultry industry (Bin *et al.*, 2007) besides having a negative repercussion on animal welfare. According to Balog *et al*. (2003) and Pavlidis *et al*. (2007), mortality due to Ascites varies from 5% to 8% in various populations, increasing up to 30% in heavier broiler flocks.

Indicators of Ascites Syndrome

Many researches are aimed on identifying definitive diagnostic indicators for AS in broilers. Right ventricle and ventricle weight ratio, hematocrit, erythrocyte counts, saturation level in the blood, heart rate, weight gain are suggested as indicator traits to be use in selection index to reduce AS susceptibility.

Hematocrit is a marker for elevated rate of erythropoiesis in ascitic birds which increases blood viscosity and results in increased resistance to blood flow through the pulmonary blood vessels. Therefore it is always significantly higher in AS broilers than in their healthy counterparts reared under the same conditions (Luger *et al.*, 2001). Heart rate obtained by pulse oximetry or by encephalography, is recorded to be lower in broilers suffering from AS than in healthy ones (McGovern *et al.*, 1999). A slower heart rate (bradycardia) as well as reduction in the pulse rate has been found in birds developing AS and in acutely cold-exposed birds (Olkowski and Classen, 1998).

Birds with high levels of oxygen saturation level in the blood (SaO_2) are less vulnerable to AS and sudden death syndrome. Selection strategy aimed at removing individuals and families that are found below the average levels of SaO_2 has led to an elevation in the SaO_2 levels of the blood, thus reducing the susceptibility of AS. At the age of 6 weeks, broilers with AS were observed to have a significantly lower SaO_2 than their healthy counterparts (62.1 and 86.0%, respectively) (Julian and Mirsalimi, 1992). As compared to healthy ones and non-AS broilers, broilers with AS induced by a pulmonary artery clamp had a significantly subservient SaO_2 and higher right-ventricle and total-ventricle weight ratio (hypertrophy of the right ventricle RV: TV) (Wideman and Kirby, 1995). Therefore, low SaO_2 was suggested to be a reliable genetic early indicator for AS vulnerability (Druyan *et al.*, 1999).

Factors responsible for Ascitis

Genetic, management, environmental, nutritional and anatomical factors all seem responsible for Ascites Syndrome have been discussed as under:

Genetic factor:

Developing a breeding aim to enhance body weight and to diminish Ascites susceptibility in broilers is of ongoing interest, because the AS is still a major challenge for poultry breeders. It has been found that heavier broilers, especially male broiler lines, are more susceptible to have AS due to intensive selection on growth rate (Decuypere *et al.*, 2000 and Pakdel, 2004). Combining low FCR with fast growth rate yields higher incidences of AS in contrary to broilers with either slower growth or higher FCR. This low FCR in fast-growing birds is responsible for low values of heat production. Birds selected for fast growth and low FCR together have low partial pressure of O_2 and high partial pressure of CO_2 in venous blood at low ambient temperature compared with the slower growing birds (Decuypere *et al.*, 2005). Ascites related traits are significantly predisposed by maternal genetic effects (Pakdel *et al.*, 2002; Navarro *et al.*, 2006 and Closter *et al.*, 2009). Therefore, not paying attention to maternal genetic effects in the model of analysis tends to overestimate direct additive genetic variances and their corresponding heritability for Ascites related traits. Various studies have reported propensity to evolve AS to be under genetic control, with estimates of heritability ranging from 0.1 to 0.7 (Lubritz *et al.*, 1995; de Greef *et al.*, 2001; Moghadam *et al.*, 2001 and Druyan *et al.*, 2007).Genetic correlations among body weight and Ascites related traits in both normal and cold

conditions have been reported (de Greef *et al*., 2001; Moghadam *et al*., 2001; Pakdel *et al.*, 2005a). Moghadam *et al*. (2001) reported a positive genetic correlation between ascites and body weight under normal condition. de Greef *et al*. (2001) and Pakdel *et al.* (2002) have shown a negative inferred genetic correlation among ascites related traits and body weight under cold condition. However, a low but positive genetic correlation between traits related to ascites measured under cold conditions and body weight measured under normal conditions was reported by Pakdel *et al*. (2005b). Moghadam *et al.* (2001) observed that the heart defect (i.e. including pulmonary hypertension, right ventricular failure and fluid accumulation in the peritoneal cavity) as a trait related to ascites is heritable and had a positive genetic correlation with body weight.

Keeping in view the development and application of genomic tools, the AS genes seem likely to be detected and mapped in the near future. Breeders will be able to easily detect and cull individual birds, within the elite lines, that carry the alleles for AS vulnerability.

Mangement and Environmental Factors:

Altitude

When birds are subjected to low atmospheric O_2 levels increasing altitude, pulmonary blood vessels constrict and pulmonary vascular resistance increases (Wideman, 1997). This immediate increase in pulmonary arterial pressure results in right ventricular hypertrophy and eventually Ascites Syndrome (Luger *et al.*, 2003). In addition, hypoxemia leads to supplement in hematocrit, which, in turn, increases blood viscosity and finally results in increased resistance to blood flow through the pulmonary blood vessels (Fedde and Wideman, 1996 and Shlosberg *et al.*, 1996). In this situation, it is vital to ventilate suitably and provide as much oxygen to the flock as possible.

Low Temperature

Low temperatures tend to escalate Ascites by increasing both metabolic O_2 requirements and pulmonary hypertension (Stolz *et al.*, 1992) which is credited to a cold induced increase in cardiac output, rather than to hypoxemic pulmonary vasoconstriction (Wideman and Tackett, 2000). Maintaining optimum brooding temperatures is important for the prevention of Ascites.

Light

It is observed that restricting the number of hours of light will slow growth slightly and will reduce activity that requires supplementary oxygen, and may actually improve feed efficiency. Further researches on the effect of extensive dark periods or intermittent lighting indicated that, similar to feed restriction, photoperiod manipulations can decline the frequency of AS (Hassanzadeh *et al.*, 2003).

Air Quality and Ventilation

Poor ventilation can induce low environmental oxygen or high toxic fumes (carbon monoxide, carbon dioxide or ammonia), which may have damaging effects on the respiratory or cardiovascular systems of birds and endorse Ascites development (Wideman, 1998). Correct litter management in combination with suitable ventilation helps to retain air quality.

Nutritional Factors:

Major nutritional factors like high nutrient density rations and feed form are known to influence the occurrence of ascites in broilers (Bolukbasi *et al.*, 2004 and Ozkan *et al.*, 2006). Besides, feed restriction minimizes growth at a

decisive time in a broiler chick's lifecycle when it is the most liable to metabolic disease due to its elevated oxygen demands (Balog *et al*., 2000 and Ozkan *et al*., 2006, 2010).

Feed restriction

It is better to prevent Ascitis than going for treatment and feed restriction is an important step in this direction. However, it curtails the growth performance, reduction of the availability of nutrients and pigmentation precursors, which may have a direct consequence on weight gain, muscle mass and the profit-cost relationship. Thus feed restriction can become a successful instrument in plummeting the incidence of Ascitis only in concurrence with good management practices.

Nutrient density

Shrinking the nutrient density can reduce the growth rate, with the effects visible from 0 to 21 days of age, during the time when birds cannot accustomed to intake lower feed nutrient content. The outcome of nutrient density on growth rate is relatively little unless the decrease in density is very large if diets is balanced to energy content. But even moderately lower nutrient density decreases mortality due to Ascites (Camacho-Fernandez *et al*., 2002).

Feed form

In the first four weeks, feeding of mash feeds rather than pelleted feeds can minimize the incidence of Ascitis without fall in market parameters.

Omega-3 Fatty Acid Sources (Flax and Fish Oils)

They seem decrease erythrocyte deformation (Bond *et al*., 1996). The increased content of unsaturated fatty acids results in amplified fluidity of the erythrocyte membrane thereby changing membrane functions to intesify the deformability of the erythrocytes and hence diminishing the incidence of Ascites.

Antioxidant

The inflated production of reactive oxygen in broilers susceptible to Ascites may result in the development of the disease or aggravate the disease as it occurs. The first line of defence against reactive oxygen in chickens is endogenous antioxidants such as tocopherols, glutathione, uric acid, and ascorbic acid. It has also been established that implants of vitamin E reduce mortality because of its antioxidant capacity (Bottje *et al*., 1995).

L-carnitine

Various studies suggest that free radicals may be involved in the development of Ascites (Bottje and Wideman, 1995). Known to have free radical scavenging properties (Packer *et al*., 1991), L-carnitine has beneficial effect on Ascites incidence. Besides, L-carnitine-supplemented chickens are more resistant to the occurrence of Ascites due to an enhanced cardiac output.

Anatomical and Physiological Factors:

Anatomy and physiology of the avian respiratory and cardiovascular systems are imperative in the receptiveness to Ascites (Decuypere *et al*., 2000; Hassanzadeh *et al*., 2005, 2008).

Blood

The viscosity of the blood is in correlation with the percentage of erythrocytes in the blood and the amount of hemoglobin (Hb) in red blood cells (Feddie and Wideman, 1996). The pH of blood may also control the binding of of Hb to oxygen in the lung and discharge of oxygen to the tissue, whereas a fall in the pH of blood lowers the oxygen affinity of Hb. van As, *et al.*, 2010 and Hafshejani, *et al.*, 2012 suggested the use of blood gas parameters like $C0_2$ partial pressure in venous blood and pH blood which are significant in predicting AS susceptibility in broiler chickens at 11 to 12 days of age.

Lung

Pulmonary hypertension (PH) with increased pressure in the right ventricle (Julian, 2007) is the outcome of instant contraction of pulmonary arterioles when the oxygen is amply reduced. Hence any stressful circumstance can result in incapability of the cardio respiratory system to meet oxygen demand (Luger *et al.*, 2003).

Heart

The right atrio-ventricular valve is composed of a muscle loop made up mainly of muscle fibers from the right ventricle wall in the avian heart which makes birds prone to valve insufficiency (Julian, 1993). Responding to increased workload, the right ventricle becomes hypertrophic and the valve hypertrophies along with the ventricle leading to rapidly developing valve failure and Ascites. When the birds are kept under standard brooding condition the AS-susceptible selected line show increased heart rate only between day 1 and day 7 contrary to AS-resistant broiler line with a decline thereafter toward day 17 (Druyan *et al.*, 2009).

Conclusion

Multiple factors contribute to Ascities Syndrome caused by interactions among Genetic, management, environmental, nutritional and anatomical factors furthering economic losses. Management and nutritional practices such as feed restriction, nutrient density and diet form have been undertaken to indirectly trim down the need for oxygen in order to reimburse the physiological limitations. Forced selection to achieve faster growing chickens consists of some anatomical and physiological limitations, which can effects bird health badly. Genetic selection aimed at some Ascties related traits especially if estimated in normal and cold weather conditions can help to achieve high weight gain without increasing Ascities incidences. Also genomic information is valuable for effective incorporation of selection against AS susceptibility into breeding programs of commercial broiler stocks.

References

[1] Balog, J.M., Kidd, B.D., Huff, G.R., Huff, W.E., Rath, N.C. and Anthony, N.B., 2003, Effect of cold stress on broilers selected for resistance or susceptibility to ascites syndrome, Poult. Sci., 82, 1383-1387.
[2] Bin, S., Keying, Z., Qiufeng, Z. and Cairong, W., 2007, Effects of ascites syndrome in broilers on their growth performances and the availability of energy and nutrients, Frontiers Agric. China, 1(2), 220-223.
[3] Bolukbasi, C., Guzel, M. and Aktas, M.S., 2004, The Effect of early feed restriction on ascites induced by cold temperatures and growth performance in broilers, J. Appl. Anim. Res., 26, 89-92.
[4] Bond, J.M., Julian, R.J. and Squires, E.J., 1996, Effect of dietary flax oil and hypobaric hypoxia on right ventricular hypertrophy and ascites in broiler chickens, British Poultry Science, 37(4),731-741.
[5] Bottje, W., Enkvetchakul , B., Moore, R. and McNew, R., 1995, Effect of a-tocopherol on antioxidants lipid peroxidation, and the incidence of pulmonary hypertension syndrome (ascites) in broilers, Poultry Science, 74(8), 1356-1369.
[6] Bottje, W.G. and Wideman, R.F., 1995, Potential role of free radicals in the etiology of pulmonary hypertension syndrome, Poultry and Avian Biological Reviews, 6, 211-231.

[7] Camacho-Fernandez, D., Lopez, C., Avila, E. and Arce, J., 2002, Evaluation of different dietary treatments to reduce the ascites syndrome and their effect on corporal characteristics in broiler chickens, J. Appl. Poult. Res., 11, 164-174.
[8] Closter, A..M., Van As, P., Groenen, M.A.M., Vereijken, A.L.J., Van Arendonk, J.A.M. and Bovenhuis, H., 2009, Genetic and phenotypic relationships between blood gas parameters and as-cites-related traits in broilers, Poult. Sci., 88, 483-490.
[9] Decuypere, E., Buyse, J. and Buys, N., 2000, Ascites in broiler chickens: Exogenous and endogenous structural and functional causal factors, World's Poult. Sci. J., 56, 367-376.
[10] Decuypere, E., Hassanzadeh, M., Buys, B. and Buyse, J., 2005, Further insights into the susceptibility of broilers to ascites, The Veterinary Journal, 169(3), 319-320.
[11] de Greef, K.H., Janss, L.L.G., Vereijken, A.L.J., Pit, R. and Gerritsen, C.L.M., 2001a, Disease-induced variability of genetic correlations: Ascites in broilers as a case study, J. Anim. Sci., 79, 1723–1733.
[12] Druyan, S., Cahaner, A., Bellaiche, M., Rosner, A., Shlosberg, A., 1999, Genetics of blood oxygenation, heart rate, and ECG waveforms, and their association with ascites in broilers, In: Proc. 1st European Poultry Genetics Symposium, Mariensee, Germany, p. 112.
[13] Druyan, S., Shinder, D., Shlosberg, A., Cahaner, A. and Yahav, S., 2009, Physiological parameters in broiler lines divergently selected for the incidence of ascites, Poult. Sci., 88, 1984-1990.
[14] Druyan, S., Shlosberg, A. and Cahaner, A., 2007, Evaluation of growth rate, body weight, heart rate, and blood parameters as potential indicators for selection against susceptibility to the ascites syndrome in young broilers, Poult. Sci., 86, 621-629.
[15] Fedde, M.R. and Wideman, R.F. Jr., 1996, Blood viscosity in broilers: Influence on pulmonary hypertension syndrome. Poult. Sci., 75, 1261-1267.
[16] Hafshejani, E.F., Gholami-Ahangaran M., and Hosseni, E., 2012, Study of blood cells, blood gases and thyroid hormones in broiler chickens suspected of ascites syndrome, Global Veterinaria, 8 (1), 18-21.
[17] Hassanzadeh, M., Bozorgmehri, M., Fard, B.H.J. and Decuypere, E., 2003, Beneficial effects of alternative lighting schedules on the incidence of ascites and on metabolic parameters of broiler chickens, Acta Veterinaria Hungarica, 51(4), 513-520.
[18] Hassanzadeh, M., Buyse, J. and Decuypere, E., 2008, Further evidence for the involvement of anatomical parameters of cardiopulmonary system in the development of ascites syndrome in broiler chickens, Acta Veterinaria Hungarica, 56, 71-80.
[19] Hassanzadeh, M., Gilanpour, H., Charkhkar, S., Buyse ,J. and Decuypere, E., 2005, Anatomical parameters of cardiopulmonary system in three different lines of chickens: further evidence for involvement in ascites syndrome, Avian Pathology, 34, 1-6.
[20] Havenstein, G.B., Ferket, P.R. and Qureshi, M.A., 2003, Growth, livability, and feed conversion of 1957 vs. 2001 broilers when fed representative 1957 and 2001 broiler diets, Poult. Sci.,82, 1500-1508.
[21] Julian, R.J., 1993, Ascites in poultry, Avian Pathol, 22, 419-454.
[22] Julian R.J., 2007, The Response of the Heart and Pulmonary Arteries to Hypoxia, Pressure, and Volume. A Short Review Poultry Science, 86(5), 1006-1011.
[23] Julian, R.J. and Mirsalimi, S.M., 1992, Blood oxygen concentration of fast-growing and slow growing broiler chickens, and chickens with ascites from right ventricular failure. Avian Diseases, 36(3), 730-732.
[24] Kalmar, I.D., Vanrompay, D. and Janssens, G.P., 2013, Broiler ascites syndrome: collateral damage from efficient feed to meat conversion, Vet. J., 197, 169–174.
[25] Lubritz, D.L., Smith, J. L. and McPherson, B.N., 1995, Heritability of ascites and the ratio of right to total ventricle weight in broiler breeder male lines, Poult. Sci., 74, 1237-1241.
[26] Luger, D., Shinder, D., Rzepakovsky, V., Rusa,l M., Yahav, S., 2001, Association between weight gain, blood parameters, and thyroid hormones and the development of ascites syndrome in broiler chickens, Poult. Sci. 80, 965-971.
[27] Luger, D., Shinder, D., Wolfenson, D. and Yahav, S., 2003, Erythropoiesis regulation during the development of ascites syndrome in broiler chickens: a possible role of corticosterone, J. Anim. Sci., 81, 784-790.
[28] McGovern, R.H., Feddes, J.J.R.. Robinson, F.E., Hanson, J.A., 1999, Growth performance, carcass characteristics and the incidence of ascites in broilers in response to feed restriction and litter oiling, Poult. Sci., 78, 522-528.
[29] Moghadam, H.K., McMillan, I., Chambers, J.R. and Julian, R.J., 2001, Estimation of genetic parameters for ascites syndrome in broiler chickens, Poult. Sci. 80, 844-848.
[30] Navarro, P., Visscher, P..M., Chatziplis, D., Koerhuis, A..N..M. and Haley, C.S., 2006, Genetic parameters for blood oxygen satu-ration, body weight and breast conformation in 4 meat-type chicken lines, Br. Poult. Sci., 47, 659-670.
[31] Olkowski, A.A., Classen, H.L., 1998, Progressive bradycardia, a possible factor in the pathogenesis of ascites in fast-growing broiler chickens raised at low altitude, Br. Poult. Sci., 39, 139-146.
[32] Ozkan, S., Takma, C., Yahav, S., Sogut, B., Türkmut, L., Erturun, H. and Cahaner, A., 2010, The effects of feed restriction and ambient temperature on growth and ascites mortality of broilers reared at high altitude, Poult. Sci., 89, 974-985.
[33] Ozkan, S., Plavnik, I. and Yahav, S., 2006, Effects of early feed restriction on performance and ascites development in broiler chickens subsequently raised at low ambient temperature, J. Appl. Poult. Res., 15, 9-19.

ISSN: 2348-1358
Impact Factor: 6.057
NAAS Rating: 3.77

[34] Packer, L., Valenza, M., Serbinosa, E., Starke-Reed, P., Frost, K. and Kagan, V., 1991, Free radical scavenging is involved in the protective effects of L-propionyl-carnitine against ischemia reperfusion injury of the heart. Archive Biochemistry Biophysics, 288, 533-537.
[35] Pakdel, A., 2004, Genetic analysis of ascites related traits in broilers, Ph D. Thesis, Wageningen Univ., Wageningen.
[36] Pakdel, A., Bijma, P., Ducro, B.J. and Bovenhuis, H., 2005a, Selection strategies for body weight and reduced ascites susceptibil-ity in broilers, Poult. Sci., 84, 528-535.
[37] Pakdel, A.., Van Arendonk, J..A,.M., Vereijken, A..L..J. and Bovenhuis, H., 2002, Direct and maternal genetic effects for ascites-related traits in broilers, Poult. Sci., 81, 1273-1279.
[38] Pakdel, A., Van Arendonk, J.A.M., Vereijken, A.L.J. and Bovenhuis, H., 2005b, Genetic parameters of ascites-related traits in broilers: correlations with feed efficiency and carcass traits, Br. Poult. Sci., 46, 43-53.
[39] Pavlidis, H.O., Balog, J.M., Stamps, L.K., Hughes, Jr J.D., Huff, W.E. and Anthony, N.B., 2007, Divergent selection for ascites incidence in chickens, Poult. Sci., 86, 2517-2529.
[40] Shlosberg, A., Bellaiche, M., Zeitlin, G., Yaacobi, M. and Cahaner, A., 1996, Hematocrit values and mortality from ascites in cold stressed broilers from parents selected by hematocrit, Poultry Science, 75, 1-5.
[41] Stolz, J.L., Rosenbaum, L.M., Jeong, D., Odom, T.W., 1992, Ascites syndrome, mortality and cardiological responses of broiler chickens subjected to cold exposure, Poult. Sci., 71(Suppl. 1), 4.
[42] van As, P., Elferink, M.G., Closter, A.M., Vereijken, A., Bovenhuis, H., Crooijmans, R.P.M.A., Decuypere, E. and Groenen, M.A.M., 2010, Poultry Science, 89(8),1684-1691.
[43] Wideman, R.F. Jr., Kirby, Y.K., 1995, A pulmonary artery clamp model for inducing pulmonary hypertension syndrome (ascites) in broilers, Poult. Sci., 74, 805-812.
[44] Wideman, R.. F. Jr., 1997, Understanding pulmonary hypertension syndrome (ascites), Hubbard Farms Technical Report, Walpole, NH, June, pp. 1-6.
[45] Wideman, R.F., 1998, Causes and control of ascites in broilers. National Meeting on Poultry Proceeding, 33, 56-85.
[46] Wideman, R.F. Jr. and Tackett, C.D., 2000, Cardio-pulmonary function in broilers reared at warm or cool temperatures: effect of acute inhalation of 100% oxygen, Poult. Sci., 79, 257-264.

