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ASCITES VERSUS SUDDEN DEATH SYNDROME (SDS) IN BROILER CHICKENS: A REVIEW

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ABSTRACT

The demand for animal protein is on the increase especially with continuous increase in human population. Therefore, the broiler chicken has been intensely bred and selected for high growth rate, increased feed conversion, and meat yield. However, Ascites and Sudden Death Syndrome (SDS) poses a serious challenge and had caused great economic losses in the poultry industry worldwide. Ascites syndrome (AS), also known as pulmonary hypertension syndrome (PHS) refers to a condition whereby there is fluid accumulation in the abdominal cavity which develops gradually and is often triggered by inadequate oxygen levels. Sudden death syndrome (SDS) on the other hand is a non-infectious metabolic disorder which is often characterized by sudden death of a healthy and heavy bird without discernible cause. It usually occurs suddenly within few seconds, the affected birds flip over on their backs and die. This review is meant to highlight the differences between these two metabolic disorders to enable poultry farmers identify them when they occur and to know the probable causes of mortalities in their flock.

Key words: ascites syndrome (AS), sudden death syndrome, broiler chickens, metabolic disorder.

INTRODUCTION

In many parts of the world, the poultry industry occupies a leading position among agricultural industries, as the main supplier of animal protein for human population (John , 2008).The demand for meat is increasing continuously, hence the broiler chickens has been intensely bred and selected for high growth rate, increased feed conversion, and meat yield (Decuypere *et al* , 2000,2005). Olkowski *et al* (2007) reported that the incidence of metabolic disorder is very low in well managed flocks. Among the various factors that causes discomfort which consequently results to death in meat type chickens are environmental (heat stress), management (poor hygiene and ventilation), diseases or physiological and nutrition. The influence of any of these factors in combinations or singly may predispose the birds to discomfort which may eventually results into death. Poultry farmers have often wonder why a healthy bird with no symptoms of severe illness could suddenly dropped dead despite judicious administration of all routine medications and vaccinations. Gupta, (2011), in a review, observed that excessive specialization and production requirements had placed a high demand on the metabolism of broiler chickens with the consequent emergence of a number of metabolic problems such as Ascites and sudden death syndromes which have been identified as the major causes of mortality related to fast growth (Gardiner *et al.*, 1988; Maxwell and Robertson, 1998; Bessei, 2006; Druyan *et al.*, 2009;

Huchzermeyer, 2012). Results of several research reviewed studies on metabolic disorders in broilers and have associated mortalities in broilers with Ascites and Sudden Death Syndromes (Decuypere *et al*., 2000; Wideman, 2001). However, the dividing line between these two metabolic disorders (Ascites and sudden death syndrome) need to be clearly understood by poultry farmers and researchers. Therefore this review is intended to identify the distinct differences between Ascites Syndrome (AS) and Sudden Death Syndrome (SDS) and the possible predisposing factors as well as preventive measures that could be taken.

ASCITES SYNDROME (AS)

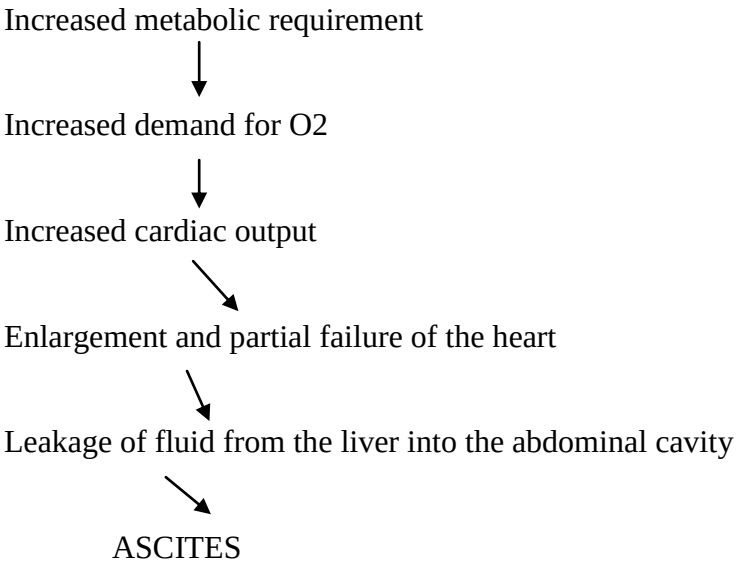
Ascites is not a disease but a multi- factorial syndrome caused by interaction between genetic, physiological, environmental and management factors (Aviagen 2009). It is a metabolic disorder characterized by increased workload of the cardiopulmonary system (hypoxaemia), central venous congestion, an accumulation of fluid in the abdominal cavity, hypertrophy of the right ventricles and a flaccid heart, all of which often lead to death (Luger *et al*., 2003;Baghbanzadeh and Decuypere, 2008; Singh *et al*., 2011;Wideman *et al.*, 2013).The condition is also known as Pulmonary Hypertension Syndrome (PHS), or water belly (Singh *et al.*, 2011, Hasanpur *et al.*, 2015). Ascites syndrome (AS) has caused huge losses to the broiler industry worldwide. It is estimated that 5% of broilers and 20% of roaster birds die of Ascites on annual basis (Balog, 2003; Bin *et al.*, 2007)).Ascites develops gradually and the birds suffer for an extended period before they die. The underlying factor to the development of these symptoms is insufficiency of oxygen supply to the tissues of rapidly growing broiler. Both genetic and environmental factors contribute to the development of the syndrome (Huchzermeyer, 2012, Wideman *et al*., 2013 and Hasanpur *et al.*, 2015). Broilers which are susceptible to ascites showed a reduced function of the tissue mitochondria (Cisar *et al.*, 2005). Environmental factors, which increase the demand for oxygen, such as low brooding temperature, or, which impair oxygen supply to the blood, such as high altitude, are known to increase the incidence of ascites (Julian, 2000, 2005).

Signs and symptoms

Generally symptoms develop when the right ventricular failure occurs. At the initial stages this can be detected by a slight darkening of the comb and wattles, as the syndrome progresses, fluid leaked out from the liver and accumulates in the abdominal cavity, leading to dilated abdomen showing pot-bellied appearance (Gupta, 2011). Consequently, breathing becomes restricted and finally results to death (Olkowski and Classen, 1999; Shome *et al*., 2000; Gupta, 2011).Because more than one organs are involved in Ascites syndrome, the symptoms are somehow complex, these includes ; generalized oedema, hydropericardium, epicardial fibrosis, lung oedema, enlarged flaccid heart, hypertrophy and dilation of the heart, hypoxaemia (Olkowski, *et al* 2003; Balog *et al*., 2003; Baghbanzadah and Decuypere, 2008;Huchzermeyer, 2012; Wideman *et al*.,2013). Report by Aviagen (2009) revealed that any factor that increases the workload of the heart by increasing the demand for oxygen can lead to Ascites.

Series of events leading to Ascites Syndrome (AS)

When the workload on the heart and lungs is increased, a chain of events is triggered that leads to reduced levels of oxygen in the blood (Lorenzoni *et al* 2006; Baghbanzadah and Decuypere, 2008).



Source: Aviagen (2009)

Causes of Ascites syndrome (AS) in broiler chickens

Environmental factor (Cold temperatures).

Cold exposure is the most important secondary factor that causes Ascites syndrome in broilers raised in an open sided and non - insulated poultry pens (Wideman and Tackett, 2000).Exposure to cold temperatures during brooding has a lasting effect on Ascites incidence (Julian, 2000; Grooves, 2002).When the ambient temperature is relatively lower than the bird's body temperature, there is a sharp increase in oxygen consumption (Huchzermeyer *et al*., 1989,Gupta, 2011). Exposure to cold periods that place the birds outside their thermo-neutral zones (10^0C-25^0C) will increase the demand for oxygen as this will force the birds to generate more energy to keep warm (Julian 1993, 2000, Decuypere *et al., 2005*).Julian *et al.* (1989); Gupta (2011) and Huchzermeyer (2012) also reported that broiler chickens reared in cold environment are more prone to incidence of Ascites. Cold environmental temperatures have the tendency of increasing tri-iodothyronine (T3) concentration, which is required for the generation of additional metabolic heat to maintain body temperature in cooler environment (Wideman, *et al.*, 2003; Gupta, 2011),.

Brooding temperature.

Lower brooding temperatures (26.7^0C, 24.4^0Cand 21.1^0C in the 1^{st}, $2^{nd}$and 3^{rd} weeks, respectively) have been shown to significantly increase Ascites mortality at 6 weeks of age, when compared with higher brooding temperatures (Huchzermeyer, 2012). Vogelaree *et al.* (1992) and Balog *et al.* (2003) had earlier reported that cold exposure causes haemo

concentration, increasing blood viscosity and blood pressure. Sato *et al.* (2002) and Ipek and Sahan (2006), gave a report that a strong correlation exist between cold temperature and Ascites. Therefore maintaining adequate brooding temperatures are critical to the prevention of Ascites. Inadequate ventilation within the pen during cold period and emission of carbon dioxide by brooders can also predispose broiler chickens to Ascites syndrome. . If chicks are exposed to cold within the first week of brooding, this may affect their metabolic rate for several weeks hence, predisposing them to Ascites syndrome (Decuypere *et al.*, *2005;*Huchzermeyer, 2012*).*

Poor ventilation

Huchzermeyer, (2012) reported that sub-optimal ventilation in broiler houses during winter leads to low environmental oxygen and higher toxic gases. Poor air quality, dust and respiratory diseases may predispose the birds to ascites as a result of respiratory damage which reduces the efficiency of respiration and blood oxygen levels (Wideman, 1998, 2001; Hassanzadeh *et al*., 2003 and Balog, 2003). Common house air contaminants that can increase ascites susceptibility includes ; ammonia, carbon dioxide, carbon monoxide, dust and humidity (Aviagen , 2009).

Contaminants	**Effect**
Ammonia	Can be detected by smell at 20ppm or above
	Above 10ppm will damage lung surface
	Above 20ppm will increase susceptibility to respiratory diseases
	Above 50ppm will reduce growth rate
Carbon Dioxide	3500ppm causes Ascites
Carbon Monoxide	100ppm reduces oxygen binding
Dust	Damage to respiratory tract lining and increased susceptibility to diseases
Humidity	Effects vary with temperature. At 29^0C and above 70% relative humidity, growth will be affected negatively.

Source: Arbor Acres Service Bulletin, December 2009.

Genetic factor

The broiler chickens has been selected for higher growth rate therefore more oxygen is required to sustain the rapid growth rate (Decuypere *et al*.,2000,2005, Gupta,2011; Huchzermeyer, 2012). The time required to reach 1.5kg live weight has been reduced from 120 days in 1925 to 30 days in 2005 (Baghbanzadeh and Decuypere, 2008).The capacity of the lungs to pump oxygen to meet this rapid growth rate is inadequate .This has been associated with impaired regulation of energy balance under extreme conditions such as low ambient temperature (Luger,*et al.* 2003).Julian, (2000) reported that if the lung of the chicken grow less rapidly than the rest of the body, it could result to hypoxia and ascites , therefore any management practices that can help to reduced growth rate will help in reducing the occurrence of Ascites syndrome.

Management

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; 2005 and Ozkan *et al*., 2006).The use of high energy or high nutrient density diets has been suggested to aggravate ascites. In addition to high energy diets pelleted feed has also been reported to increase the incidence of Ascites syndrome (Silva *et al* 1988 and Ekanayake *et al* 2004). Bolukbasi *et al*.,(2005), reported that incidence of ascites syndrome is higher in broilers that consume pellet feed than those that consume the same diet in mash form. Mycotoxins and high levels of furazolidone in feed are also potential causes of ascites in broilers (Bhagat *et al*., 1990; Richard, 1993). Bowes (1988) and Dale, (1990) reported a high incidence of ascites in 3-weeks old broiler chicks that ate feed containing a toxic level of dicalcium phosphate which resulted into lung damage with calcification in the affected birds.

SUDDEN DEATH SYNDROME (SDS)

Sudden death syndrome (SDS) is also known as morte subita, acute death syndrome, heart attack, dead in good condition, lung oedema, and flip - over disease (Saki and Hemati 2011). It is characterized by the sudden death of well nourished broiler chickens after abrupt and brief flapping of their wings (Saki and Hemati, 2011). Death usually occurs within 1-2 minutes with the birds lying on their backs with outstretched wings. According to Julian, (2005), young, healthy, fast growing broiler chickens had been observed to have died suddenly while standing, walking, sparring, or feeding. Newberry *et al* (1987),Olkowski *et al.*, (2008)also reported that SDS chickens exhibit a sudden attack which last for an average of 53 seconds prior to death., The attack is often characterized by failure of balance, violent flapping and strong muscular contraction . Usually, such birds show no outward signs of disease but suddenly extend their necks gasp or squark, flap their wings and then flip over on their backs (Kaul and Trangadia, 2003, Nain, *et al* 2007).Heavy breeds or fast growing breeds are more predisposed to SDS especially, the males. Olkowski *et al*., (2008) reported that a higher growth rate may be responsible for the high susceptibility of the males to SDS.Also, the vulnerability of broiler chicken to SDS is co-related with metabolic characteristics imposed by rapid growth rate.There may be no signs or symptoms of ill- health prior to death. It is non-infectious and often characterized by sudden death of a healthy and heavy bird without discernible cause (Olkowski *et al.*, 2008).

Causes of sudden death syndrome (SDS) in broiler chickens

Although the physiological mechanisms causing SDS have not been fully understood, the influence of diet form, texture and quality cannot be overlooked (Scott, 2002, Kaul and Trangadia, 2003; Saki and Hemati 2011). Feeding broilers on high nutrient density diet enhance rapid growth rate which consequently predispose the birds to metabolic disorder resulting to sudden death syndrome (Singh *et al*., 2011, Banday *et al*., 2011). Death has been said to occur within 37 to 69 seconds from the time the first sign of unrest is observed (Newberry *et al.*, 1987).SDS can occur as early as 3 days of age and may continue until birds reach market weight. Peak mortality usually occurs between 12 and 28 days of age (Saki and Hemati, 2011).Because there are often no specific gross or histological lesions present after post mortems data on SDS is scanty since dead birds are typically well fleshed with feed in the digestive tract and normal gall bladder as evidence that the bird was healthy before the SDS attack. (Jacquie, 2015).

Differences between Ascites Syndrome (AS) and Sudden Death Syndrome (SDS)

i.	Ascites syndrome (AS) is a multi-factorial syndrome caused by interactions between genetic, physiological and environmental (Aftab and Khan, 2005; Baghbanzadeh and Decuypere, 2008; Avigens, 2009; Singh *et al.*, 2011).	Sudden Death Syndrome (SDS) is often associated with nutrition (high density diets) and environmental (heat stress) factors according to (Saki and Hemati 2011).Thus, any problems that arise from pressure of high metabolic rate due to rapid growth can cause SDS (Olkowski and Classen, 1995; Moghadam *et al.*, 2005).
ii.	Ascites" refers to the fluid accumulation in the abdominal cavity, hypertrophy of the right ventricle resulting in flaccid heart and finally death (Baghbanzadeh and Decuypere, 2008; Singh, *et al*., 2011). It is characterized by hypoxaemia, increased workload of the cardio – pulmonary system, central venous congestion (Luger,*et al*. 2003),	SDS is a metabolic stress, caused by diet related metabolic discomfort leading to cardiac dysfunctions (Olkowski and Classen, 1995; Moghadam *et al.*, 2005; Bessei, 2006; Saki and Hemati, 2011).
iii.	Ascites syndrome is also known as pulmonary hypertension syndrome (PHS), or water belly (Singh,*et al.*, 2011;Hasanpur,*et al.*, 2015).	SDS is known as acute death syndrome, heart attack, dead in good condition, lung oedema, flip - over disease and morte – subita from a Latin word *subitus* meaning sudden (Saki and Hemati 2011).
iv.	Ascites develops gradually and the birds suffer for an extended period before they eventually succumb to death (Bessei, 2006). Inadequate oxygen levels often trigger a series of events which consequently results to AS (Aftab and Khan, 2005; Aviagen, 2009; Gupta 2011)	SDS often occurs suddenly while birds are busy with normal activities such as standing, walking, sparring, or feeding without previous signs of ill health (Newberry *et al*. 1987, Olkowski *et al*. 2008). The birds exhibit sudden attack which may not last beyond 53 seconds prior to death (Newberry, *et al*. 1987; Saki and Hemati 2011).
v.	Ascites Syndrome is heritable. (Moghadan, *et al*. 2005; Decuypere, *et al*. 2005, Olkowski, *et al*. 2007; Druyan, *et al*. 2007).	SDS is not heritable (Moghadan, *et al.* 2005; Banday, *et al*. 2011).
vi.	In the case of AS, the affected birds show signs of cyanosis, dilated abdomen and also, necropsy findings shows gross lesions (Shome, *et al.* 2000; Gupta, 2011; Huchzermeyer, 2012).	In the case of SDS, the affected birds shows no specific abnormalities and are always well fleshed with no lesion (Imeada, 2000; Saki and Hemati, 2011). However, the pathological lesions seen in SDS are associated with some type of vascular disturbance (Banday, *et al*., 2011).

Possible Preventive Measures against Ascites and Sudden Death Syndrome

Both conditions are undoubtedly related to fast growth rate and as such any management technique to reduce the early maximum genetic potential for growth can help in preventing these metabolic disorders. These preventive measures include:

i. Reducing the birds' metabolic oxygen requirement by slowing growth or reducing feed density (Wideman and Tackett, 2000, Decupere *et al.,* 2000).

ii. Maintain adequate brooding temperature (Sato *et al*. 2002, Gupta, 2011).

iii. Intermittent lighting to reduce the duration of feeding, then consequently slow down growth (Julian, 2000, Hassanzadeh *et al.,* 2000, 2003).

iv. Addition of ascorbic acids at the rate of 50mg/kg diet reduced the ascites incidence in broiler chickens (Xiang *et al*., 2002).

v. Reduce the nutrient density of the diet, using diets with 5-7% less nutrient density, thereby delay early fast growth rate (Camacho – Fermandez *et al*., 2002; Scott, 2002; Banday *et al.,* 2011).

vi. Feeding mash diets to broiler chickens to slow down growth rate as compared to when pelleted diets are fed (Madrigal, *et al* 2002, Ekanayake *et al* 2004, Bolukbasi *et al.* 2005)

vii. Practice feed restriction program that limit daily feed intake to 75% of the ME required for normal growth (Balog *et al.,* 2000; Madrigal *et al*., 2002; Aftab and Khan, 2005, Saki and Hemati, 2011).

viii. Ensure adequate ventilation and air quality in the poultry pen (Madrigal *et al*., 2002; Baloq, 2003; Aftab and Khan 2005).

ix. Feeding low chloride with high bi - carbonate diets results in a decrease in pulmonary hypertension (Squires and Julian 2001).

CONCLUSION

Within the scope of this review, it is concluded that Ascites syndrome (AS) and sudden death syndrome (SDS) are not diseases but rather metabolic disorders often triggered by interactions between genetics, environmental and nutritional factors. Ascites syndrome is characterized by excessive accumulation of fluid in the abdominal region whereas Sudden death syndrome (SDS) is characterized by sudden death of an apparently healthy bird without prior sign of ill health.

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