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## **New Approach for the Incidence of Ascites Syndrome in Broiler Chickens and Management Control the Metabolic Disorders**

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**Abstract:** Metabolic diseases such as ascites in broiler chickens result in significant economic losses to the poultry industry. The syndrome is multifactorial and mainly caused by exogenous and/or endogenous factors. Occurrences among faster growing lines are even more pronounced under conditions that imposed an additional metabolic load on the birds such as low ambient temperature. So, particular interactions between the environmental as well as with the genetic factors, play an important role. The purpose of this paper is to review some of predisposing factors that increase ascites incidence and the preventive procedures that have emerge for reducing the incidence of metabolic disorders in broiler chickens. Additionally, spontaneous hypoglycaemia or spiking mortality syndrome is also briefly mentioned.

**Key words:** Ascites, SDS, spiking mortality, chickens, lighting, feed restriction

### **INTRODUCTION**

During the last decades, several livestock species, particularly meat type chickens have been intensively selected for improved growth rate. This has greatly reduced the time to reach the desired market weight. However, increased growth rate is accompanied by a severe incidence of ascites in the commercial broiler industry throughout the world (Olkowski and Classen, 1999, Hassanzadeh *et al.*, 2005ab, 2008). Ascites is a serious economic concern because it results in the losses of the heaviest and fastest growing broilers. In the World Broiler Ascites Survey (Maxwell and Robertson, 1997), information on 18 countries from four continents showed that ascites affects on average 4.7% of all live broilers worldwide. The causes of the syndrome are multifactorial and mainly induced by exogenous and/or endogenous factors. An imbalance between oxygen supply and the oxygen required to sustain rapid growth rates and high food efficiencies is believed to be the primary cause of ascites in broiler chickens (Decuypere *et al.*, 2000, 2005; Julian, 2005). Insufficient development anatomical parameters of the lungs or the pulmonary blood vessels in broiler chicken lines may form the basis of ascites incidence. Changes in haematocrit values, partial pressure of oxygen and carbon dioxide and the endocrine functioning, such as thyroid hormone activity, are also important regulatory mechanisms of metabolic rate during the embryonic development and growing period (Buys *et al.*, 1998a; Decuypere *et al.*, 2000, 2005; Hassanzadeh *et al.*, 2002, 2004, 2005ab, 2008). Thyroid hormone activity for regulating metabolism could become more apparent at different genotype of chickens (Decuypere *et al.*, 2000; Luger *et al.*, 2001) and different lighting programmes (Buyse *et al.*, 1994; Buys *et al.*, 1998b; Hassanzadeh *et*

*al.*, 2000, 2003) and even at high altitude in broiler chickens (Hassanzadeh *et al.*, 2002, 2003, 2004, 2005ab, 2008). Indeed, in cases that a decrease in thyroid hormones activity may lead to mismatch between oxygen delivery and the oxygen demanding, resulting in anoxia, hypoxemia a hypoxia.

The purpose of the present paper is to evaluate the endogenous factors and environment interaction as predisposing factors for development of ascites syndrome. Furthermore, particular attention will be paid on management procedures such as different lighting schedules, feed restriction and vitamin supplementation for reducing the incidence of metabolic disorders such as ascites and SDS by altering e.g. the metabolic load on broiler chickens. Additionally, spontaneous hypoglycaemia or spiking mortality syndrome is briefly discussed.

### **New approach for the incidence of ascites syndrome in broiler chickens**

**Endogenous and environmental factors interaction in ascites susceptibility:** Ascites is a metabolic disorder that is no longer restricted to high altitude (Albers and Frankenhuis, 1990) as the last decades similar signs in broiler chickens were observed at sea level (Scheele and Frankenhuis, 1989; Julian, 2000; Hassanzadeh *et al.*, 2001).

In acute hypoxia the increase in cardiac blood flow may contribute to the delivery of oxygen to the various tissues (Nesarajah *et al.*, 1983). Olkowski and Classen (1999) found that ascites susceptibility of fast growing broiler chickens at sea level was associated with bradycardia, which is the opposite of tachycardia at high altitude. They argued that a low cardiac output could be the haemodynamic key problem leading to cardiovascular failure in fast growing broilers.

Many authors reported that a low thyroid hormone activity was accompanied in chickens selected for rapid growth rate and improved feed efficiency (Decuyper *et al.*, 2000) that could also be related to ascites susceptibility in broiler chickens (Scheele *et al.*, 1992, 2003; Decuyper *et al.*, 2000; Luger *et al.*, 2001; Malan *et al.*, 2003). Authors argued that decrease thyroid hormone will play a significant role in the inability to provide adequate oxygen delivery in chickens that lead to anoxia, heart failure and ascites.

Regarding the Malan *et al.* (2003) thyroid hormone deficiency could also impair lung development and therefore could be elevated the risk of ascites incidence in rapid growth chickens. Additionally, Hassanzadeh *et al.* (2005b, 2008) documented a direct correlation between lungs development of post hatch broiler chickens with the embryonic thyroid hormones activities. These investigations showed that post hatched chickens had higher lungs volumes in relation to their body size when had higher thyroid hormones concentrations during the embryonic development (Fig. 1). Consequently, such chickens with higher lungs volume demonstrated less susceptible to incidence of ascites when exposed to chronic hypoxia at high altitude. Ascites susceptibility in fast growing broiler chickens is not attributed to high oxygen requirement per se but rather to an imbalance between oxygen demands and oxygen supply, resulting in anoxia, hypoxemia and hypoxia (Decuyper *et al.*, 2000). The imbalance of oxygen requirements and supply may be caused either by an extremely high metabolic demand by the tissues (anoxia) or by an insufficient supply of oxygen (hypoxia), or both. This imbalance in both sides can be caused by exogenous as well as by endogenous factors, and both sets of factors can potentiate the other, acting in an additive or even in synergistic way (Decuyper *et al.*, 2000; Decuyper, 2002).

**Embryonic development and ascites incidence:** The peak of ascites incidence occurs in weeks 5-6 of the growing period, but it is thought that the aetiology of the syndrome is initiated much earlier, even during the embryonic stage (Coleman and Coleman, 1991). As the chick embryo consumes 60% more oxygen between the start of pulmonary breathing and hatching compared to earlier stages (Visschedijk, 1968), it is possible that a shortage of oxygen occurs during this stage. A reduction in the duration of subsequent prenatal and perinatal period might reduce this hypoxic situation. Dewil *et al.* (1996) showed that ascites resistant broilers hatched earlier than more sensitive ones, and that this is linked to the higher thyroid activity in the resistant line. A high carbon dioxide concentration in the air chamber is also trigger for hatching (Decuyper, 2002). The findings of Chineme *et al.* (1995) indicated that the length and/or severity of prenatal hypoxia may influence postnatal

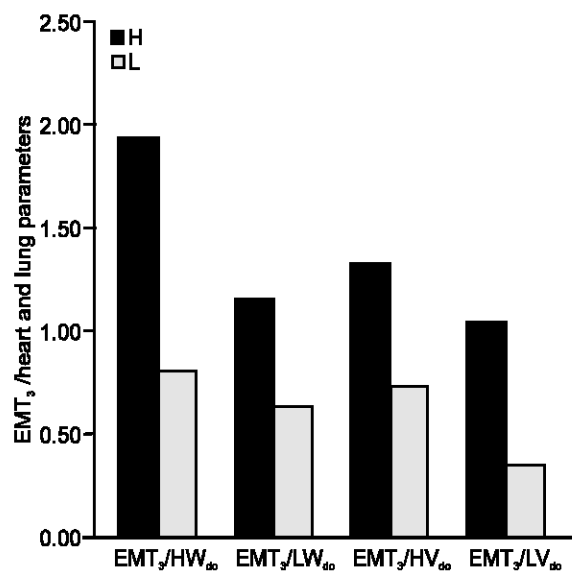


Fig. 1: The ratios of embryonic T3 levels to Heart Weight (HW), Lung Weight (LW), Heart Volume (HV) and Lung Volume (LV) of Day-old Broiler (do) chickens that hatched at High (H) or Low (L) altitudes (Hassanzadeh *et al.*, 2008)

characteristics related to ascites. Eggs incubated in an environment with a high concentration of carbon dioxide during the last week (Buys *et al.*, 1998a; Hassanzadeh *et al.*, 2002) and/or first 10 days of incubations (Decuyper *et al.*, 2006; De Smit *et al.*, 2005, 2006) hatched earlier than in an environment with normal CO<sub>2</sub> levels (Fig. 2). Furthermore, embryos incubated at high altitude, 2000 m above sea level, had earlier hatching time than those incubated at low altitude (Hassanzadeh *et al.*, 2004). In these studies those chickens which hatched earlier showed a lower incidence of ascites during the growing period because, high concentrations of carbon dioxide in the incubation environment might ironically decrease the length of time the embryo experiences hypoxia (Buys *et al.*, 1998b; Hassanzadeh *et al.*, 2002, 2004; De Smit *et al.*, 2006). The severity of embryonic hypoxia may be related to porosity and structure of the egg shell and hence, to the partial pressures of oxygen and carbon dioxide in the egg and air chamber, especially during the last days of incubation (Decuyper, 2002). Sadler *et al.* (1954) explained that these beneficial effects of carbon dioxide were the result of the reduction of pH of the albumen which might have retarded the apparent breakdown of the chalaziferous membrane and to the thick layer of albumen and leading to reduce the of length incubation. Alternatively, it could be related to the duration of this hypoxic period and hence, to the pipping and hatching time.

De Smit *et al.* (2006) mentioned that increase in carbon dioxide concentration in the first 10 days of incubation

seems to switch on some physiological systems related to embryonic development earlier than it happens under normoxic conditions or changes the rate of these processes. Blacker *et al.* (2004) provided evidence for the role of hypoxia during the embryonic development in the control of pulmonary surfactant (Fig. 3). Authors suggested that the avian pulmonary surfactant system exhibits a high level of plasticity within the early stages of surfactant maturation. Possible explanation is an interaction between environmental and endogenous factors e.g corticosterone and thyroid hormones, during the critical development of chick embryos (Hassanzadeh *et al.*, 2004; Blacker *et al.*, 2004) that exploit developmental plasticity, resulting to physiological heterokairy of surfactant system by altering both the rate and onset of development of the surfactant lipids and earlier commencement of air breathing. These observations indicate that hypoxic conditions occurred in the embryonic can alter the developmental trajectories of some endogenous parameters in prenatal and postnatal chicks. The development of these important parameters is favourable to increase gas exchange area and results to lower susceptibility of birds to pulmonary hypertension and may be a predisposing factor for later development of heart failure and ascites syndrome. Hypoxic conditions during embryonic development may initiate structural changes in the cardiovascular and pulmonary systems, as was observed in our recent study at high altitude (Hassanzadeh *et al.*, 2005b, 2008). In these studies 1-day-old of high altitude hatched-chicks showed significantly higher proportional heart ( $1.23 \pm 0.08$  versus  $0.87 \pm 0.07$ ) and lung ( $2.02 \pm 0.07$  versus  $1.24 \pm 0.13$ ) weights to body weight compared to low altitude hatched-chicks, while no significant difference was found at slaughtered age, when birds were reared at high altitude. This coincided markedly with reduced incidence of ascites mortality and right ventricular hypertrophy in high altitude hatched-birds than to the low altitude hatched-birds (Hassanzadeh *et al.*, 2004, 2008). In these studies changes of anatomical parameters were accompanied with the increase of hormonal activity of embryos, demonstrating critical role of endogenous parameters on maturation of pulmonary system (Fig. 1 and 2). Therefore, this early stages of development may be decisive in determining the subsequent adaptive capacity to cope with unfavourable conditions after hatching (Decuypere, 2002, 2006; Hasanzadeh *et al.*, 2004, 2008).

**Management control of metabolic disorders**

**Lighting schedules and ascites:** Domestic fowl are photoperiodic nibblers by nature, meaning that they consume feed continuously throughout the entire daylight period. Commercial broiler chickens are reared under Continuous Lighting (CL) because it is believed that feed intake is then maximal and hence their growth

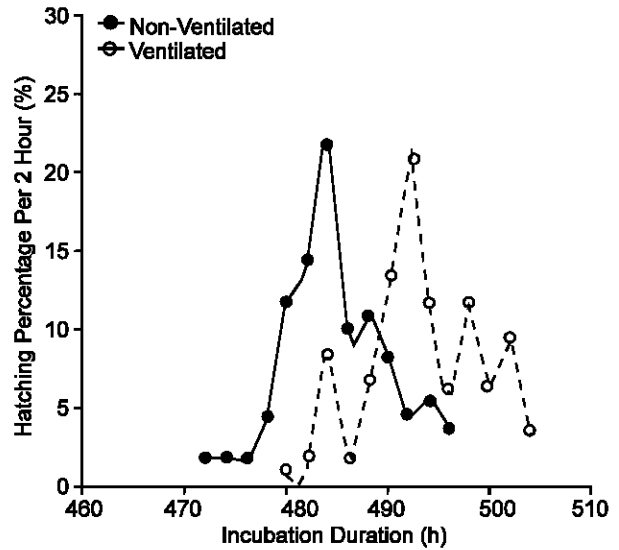


Fig. 2: Comparative percentage of hatched chicks of the non-ventilated and ventilated incubator (Decuypere *et al.*, 2006)

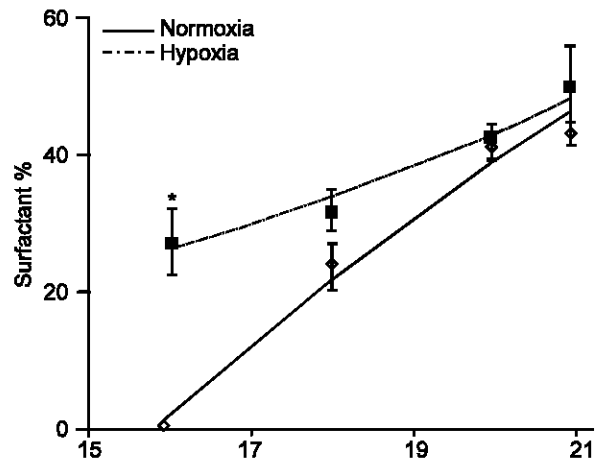


Fig. 3: Comparative percentage of pulmonary surfactant in normoxia and hypoxia chick embryos (Blacker *et al.*, 2004)

rate (Buyse and Decuypere, 2003). However, there are many disadvantages to constant lighting programmes as metabolic disorders such as leg problems, sudden death syndrome and ascites are much more common (Buyse *et al.*, 1996). Several studies, have indicated that providing birds with an Intermittent Lighting Programme (IL) is beneficial because these alternative lighting programmes not only improve broiler performance but also reduce the occurrence of metabolic disorders by controlling the rate of early growth of the young chickens. Fast-growing broiler chickens are characterized by a very high specific metabolic rate at about 2 weeks of age (Buyse *et al.*, 1994). This high metabolic rate requires

high oxygen needs and hence make those fast-growing broiler chickens very susceptible for ascites development. Furthermore, malfunctioning of critical organs such as the heart and lungs is directly linked with the ascites syndrome (Decuypere *et al.*, 2000; Julian, 2000; Hassanzadeh *et al.*, 2005b). The manipulation of lighting schedules during the starter period is proven to be a valuable method that reduces the losses due to ascites in broiler chickens. Imposing intermittent lighting schedules (1L:3D) during the 24 h (Buys *et al.*, 1998a) or during the 12 h of natural scotoperiod, from 8 p.m to 8 a.m (Hassanzadeh *et al.*, 2000) reduced significantly the incidence of right ventricular failure and ascites in broiler chickens. In both studies the change of CL to IL at an early age was followed by initial growth depression as described earlier (Buyse *et al.*, 1994, 1996). This depression was, however, followed by a period of compensatory growth, in a way that the birds reared in IL reached the same final body weight by 6 weeks (Buys *et al.*, 1998a,b) and 7 weeks (Hassanzadeh *et al.*, 2000) of age. IL resulted in an overall decreased FCR during the 6 weeks of growing period (Buys *et al.*, 1998a), but such result was not observed during 7 weeks of age (Hassanzadeh *et al.*, 2000). This difference could be due to effect of genotype, nutrition and environmental interactions as reported earlier in broiler chickens reared under intermittent lighting (Buyse *et al.*, 1996; Buys *et al.*, 1998a). In both studies the same underlying physiological mechanisms have been argued. These include the lower heat production and consequently lower oxygen consumption in IL birds during the dark period of each light : dark cycle (Buyse *et al.*, 1996). Additionally, a second mechanism is the altered growth pattern between IL and CL chickens. Indeed, change from CL to IL at a young age slows down juvenile growth rate, hence the high oxygen requirements and in this way, the predisposition for incidence of ascites is alleviated. The reduction in juvenile growth rate with a concomitant reduction in metabolic rate at a young age to imposing IL was also reflected in several physiological parameters. Hematocrit as well as plasma concentrations of the biologically active thyroid hormone triiodothyronine were also lower in juvenile IL chickens compared to that of their CL counterparts.

Recent studies again confirmed the effect of alternative lighting schedules on the incidence of ascites in broiler chickens. When performed at high altitude, (2000 m above sea level), the same results were found when the intermittent lighting schedule (1L:3D, repeated six times daily from 3-42 days) or increasing photoperiod schedule (4-14 days, 6L:18D; 15-21 days, 10L:14D; 22-28 days, 14L:10D; 29-35 days, 18L:6D; 36-42 days, 23L:1D) were used for reducing of the incidence of ascites in broiler chickens (Hassanzadeh *et al.*, 2005a). The beneficial effect of intermittent lighting on the

incidence of ascites was more pronounced when IL was only applied from day 3-14 and/or from day 10-21 of age (Hassanzadeh *et al.*, 2003). In these two studies, the reduction of ascites mortality also coincided with a temporary reduction in growth rate at young age, with a concomitant reduction of metabolic indicators e.g. haematocrit values and plasma thyroid hormones concentrations, both indicating reduced of oxygen utilization and heat production.

**Antioxidant vitamins and ascites:** Vitamin C, or L-ascorbic acid, is a water-soluble vitamin widely distributed in plants and animals. It is of major importance in nutrition to maintain a good health status. Generally, ascorbic acid is not regarded as a dietary requirement for poultry because it can be synthesized at a sufficient rate to meet the needs under normal conditions. May and McNaughton (1980) could not demonstrate a positive effect of 0.1% ascorbic acid supplementation on body weight of broiler chickens and did not found any affects on thyroid hormone functions. However, dietary vitamin C has been reported to improve resistance to a variety of stressors including environmental (e.g. heat stress), nutritional and pathological conditions (Agudelo, 1983). There are several reports (Agudelo, 1983; Enkvetchakul *et al.*, 1993) describing that free radical mediated mechanisms may be involved in the etiology of pulmonary hypertension and ascites. The production of free radicals is enhanced by systemic hypoxia, inflammation and thyroid hormones (Bottje and Wideman, 1995). The potential of oxygen-derived free radicals for cytotoxic damage was also proposed by Maxwell *et al.* (1994). They also argued that oxygen free radicals causing tissue damage through lipid peroxidation of cell membranes lead to increased membrane permeability. Bottje and Wideman (1995) suggested, vitamin C is an antioxidant that can reduce oxygen-derived free radicals in which play an important role in the genesis of tissue damage during inflammatory reactions in ascitic birds.

Supplementation of 500 ppm vitamin C significantly reduced mortality due to ascites in broiler chickens with a concomitant reduction in plasma thyroid hormone levels, suggesting a role of vitamin C in metabolic activity (Hassanzadeh *et al.*, 1997a). Julian (1993) reported that the reduction of ascites in vitamin C treated birds could be a consequence of the reduction of blood flow resistance, especially in the narrow capillaries of the lungs. This facilitation of blood flow may be due to a changed packed cell volume, however no major effect of Vitamin C on haematocrit values was found in the study of Hassanzadeh *et al.* (1997ab). Xiang *et al.* (2002) reported a reduction in the incidence of pulmonary hypertension of cold-stressed and T<sub>3</sub>-fed broiler chickens when vitamin C was supplemented to their

diet. This was due to a reduced muscularisation of pulmonary arterioles of the vitamin supplemented chickens.

Vitamin E, or  $\alpha$ -tocopherol, another antioxidant, has a high lipid solubility and is located in plasma and organel membranes such as mitochondria. Vitamin E is essential in maintaining the integrity of the cardiovascular and other systems. It is considered the major chain breaking antioxidant that scavenges oxygen free radicals and prevents further peroxidative damage of cell membranes (Bottje and Wideman, 1995; Lorenzoni and Ruiz-Feria, 2006). There are reports that indicate a protective role of vitamin E in lowering pulmonary hypertension syndrome and ascites mortality through improved tissue antioxidant capacity (Enkvetchakul *et al.*, 1993; Bottje and Wideman, 1995; Lorenzoni and Ruiz-Feria, 2006). So, the antioxidant properties of oxidized vitamin E can also be restored by vitamin C. As vitamin C is able to donate an electron to the tocopherol radical generating the reduced antioxidant form of vitamin E, suggests that a major function of vitamin C is to recycle the vitamin E radical (Bottje and Wideman, 1995).

**Feed restriction and ascites:** In last two decades, there has been much emphasis on temporary food restriction of young broilers as a means to improve zootechnical performance. Studies have shown that compensatory growth, improved feed conversion ratios and reduced fat content occur in previously feed-restricted birds during the final growing period (Shlosberg *et al.*, 1991; Gonzales *et al.*, 1998). There are also reports that the incidence of ascites was significantly reduced with no adverse effect on flock performance (Albers *et al.*, 1990). However, the possibility of a lower final body weight reflected in lower breast muscle weight has been the main drawback of applying of food restriction programmes mainly due to insufficient compensatory growth after restriction (Arce *et al.*, 1992; Balog *et al.*, 2000). Food restriction by limiting of the time of *ad libitum* food intake was aimed at reducing of ascites without compromising body weight (Albers *et al.*, 1990; Balog *et al.*, 2000). Skip-1-day food restriction programmes during early growth effectively reduce ascites (Arce *et al.*, 1992). It has been experimentally shown that factors such as age at initiation of restriction, duration of restriction and its severity determine to a large extent the ability to fully manifest compensatory growth (Zubair and Leeson, 1996; Lippens *et al.*, 2000; Camacho *et al.*, 2004; Julian, 2005). Food restriction should not be started too late, for a not too long period and surely not too severe, otherwise catch-up growth is not fully manifested (Buyse and Decuyper, 2003). Other interacting factors are broiler strain and lines within strains and sex (Lippens *et al.*, 2000; Decuyper *et al.*,

2000). The earlier an effective food restriction programme can be introduced the smaller the potential negative effect on final body weight at market age (Julian, 1993).

**Sudden Death Syndrome (SDS):** SDS is a disease known as "acute death syndrome" or "Flip-over disease", is characterized by an acute death of well-nourished and seeming healthy broiler chickens after abrupt and brief flapping of their wings (Ononiwu *et al.*, 1979; Hulan *et al.*, 1980). This syndrome represents a major economic loss to the broiler industry and its occurrence of SDS is about 0.5-5% in broiler chickens and peaks in chickens at about 2-4 week of age (Summers *et al.*, 1987). A variety of factors, including of growth rate, vitamins, fats in the diet, pelleting of feed and lighting have been suggested to influence the incidence of SDS, but the etiology of SDS remains unknown (Hulan *et al.*, 1980; Gonzales *et al.*, 1998; Olkowski, 2007).

According to pathological observations, SDS is frequently, but not exclusively, accompanied by some failure of the cardiovascular system, ventricular arrhythmias and catastrophic ventricular fibrillation (Olkowski and Classen, 1997) myocardial degeneration, atherosclerotic changes of the coronary arteries and myocardial necrosis (Ononiwu *et al.*, 1979; Kawada *et al.*, 1994). Therefore, SDS has been suggested to be a cardiac disease. Biochemical data for SDS are very limited, because the lack of symptoms makes it difficult to collect samples tissues or fluid samples from SDS birds without additional post-mortem changes. A study of Imaeda (1999) shows that the serum level of enzymes utilized as indicators for clinical diagnosis of human circulatory disturbance such as Lactate Dehydrogenase (LDH) and Glutamic Oxaloacetic Transaminase (GOT) are elevated in association with SDS.

It is obvious that there is no single treatment or preventive system for the control of SDS in young broiler chickens. The condition is undoubtedly related to fast growth rate and as such, management techniques to reduce the early maximum genetic potential for growth offer the best preventative scenario. Although arrhythmias associated with outright vitamin or mineral deficiencies are rather unlikely to occur in modern broiler flocks, the risk of arrhythmia and SDS may be increased when some nutrients are supplemented in excess (Nain *et al.*, 2006).

A temporary reduction in initial growth rate achieved by reduction in daylength, or the use of low-nutrient diets, or quantitative feed restriction 20% restriction from day 8-21 (Gonzales *et al.*, 1998) can reduce the incidence of SDS with the possibility of partial or even complete growth compensation by 42 or 49 days, respectively (Decuyper *et al.*, 1999).

### **Hypoglycemia/Spiking Mortality Syndrome (HSMS):**

Spiking mortality syndrome of chickens is characterized by sudden increases in mortality for at least 3 consecutive days with low morbidity (Davis *et al.*, 1995). The affected birds are hypoglycaemic, with blood glucose levels of less than 180 mg/dl. The normal range is  $\pm$  240 mg/dl while fasting glucose is 150 mg/dl (Davis, 1997). The general symptom of spiking mortality in broilers is a sudden and unexpected increase in mortality from 7-14 days of age. As the condition progresses chicks are found in lateral or sternal recumbency with one or both legs stretched out behind them. Clinical signs include huddling of the birds, trembling, blindness, loud chirping, litter eating, ataxia, comatose, birds dead with breast down and feet and legs straight out behind birds. Males are predominantly affected and survivors exhibit great variation in sizes. Lesions described with this syndrome include hemorrhages in the liver with necrosis of liver cells, regressed thymus, regression of the bursa of Fabricius, dehydration with the accumulation of kidney urates, fluid in the crop, fluid in the lower gut and watery contents of the ceca. (Davis *et al.*, 1995; Davis, 1997). Death often occurs within two to six hours after the onset of the symptoms. Characteristically, birds that exhibit clinical symptoms but survive the acute phase, will continue to be unthrifty and stunted for the rest of the grow-out period. However, the etiology of spiking mortality is unknown but there have been a number of different causative agents suggested e.g. viral infectious (Davis, 1997), nutritional factors such as toxins in rendered product and mycotoxins (Brown *et al.*, 1991). Management aspects including of lighting programs, feed composition, feeder type and environmental conditions also appear to contribute to the problem (Davis *et al.*, 1995; Davis, 1997).

A number of management techniques mainly based on the stockmanship of broiler farmers rather than on scientific have been developed to reduce the chance of HSMS. Collecting acute suffering birds from the flock and putting them in darkness without feed seem to promote the recovery of these birds. Other remedies tried have included, changing feed, vitamin supplementation in the water, and adding sugar to the drinking water. No consistent success have been achieved with these treatments. However, the physiological basis of this metabolic disease as well of the mechanisms involved in the recovery is unclear at present. One of the mechanisms might be an endocrine disturbance. In view of the pronounced hypoglycaemia, an aberration in insulin:glucagon ratio as primary regulators of carbohydrate metabolism can be inferred (Davis *et al.*, 1995). Furthermore, plasma levels of Insulin-like Growth Factor-I (IGF-I) are depressed in HSMS turkeys (Davis *et al.*, 1997) and chickens (Buyse, personal communication). Whether this phenomenon is

related to a changed growth hormone (primary modulator of IGF-I production) metabolism is yet speculative.

Finally, putting HSMS chickens in darkness enhances recovery and this might be related to increased production and secretion of melatonin. This pineal hormone may help to restore glycemia in a direct way or indirectly by growth hormone. These hormonal aspects are currently under investigation.

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