

Does Nutrition Help to Alleviate Sudden Death Syndrome in Broiler Chicken? An Overview

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Abstract: Problems in body metabolism caused various metabolic disorders in broiler and the cause of the large proportion of morbidity, mortality and economic losses in commercial production. Metabolic diseases result mainly from rapid growth, high nutrient intake, or high metabolic rate. Cardiovascular diseases, ascites and sudden death syndrome in broilers are important types of metabolic disorders. Sudden death syndrome (SDS) is the name predicate to death in healthy fast-growing commercial broilers that are in good condition die suddenly. SDS is a major problem in the broiler industry in many parts of the world and has caused major economic losses in the broiler industry worldwide for many years. Broilers are affected at very early ages and continuing the whole time to market age. A variety of nutritional, environmental and management factors and genetics have been suggested to induce SDS. Nutritional factors are very important to attain genetic potential and prevention of metabolic disorder. Therefore, in this overview, the role of nutritional factors was considered in this regards. Based on this review the role of nutrition manipulations was found to be very low and problems occurred by incorrect selections or managements can not prevent by nutritional strategies.

Key words: Sudden death syndrome • Broiler • Nutrition

INTRODUCTION

Because of advances in genetic selection, management and nutrition, the modern-day old commercial broiler chickens have high growth and metabolic rates which make broilers highly susceptible to heart-related diseases. Sudden death syndrome, (known as *morte subita*, acute death syndrome, dead in good conditions, heart attack, lung oedema, or flip-over disease) is a condition commonly observed in fast-growing boiler chickens, whereas suggested apparently normal, healthy and heavier individuals die suddenly of an indiscernible cause [1]. It is characterized by the sudden death of well-nourished broiler chickens after abrupt and brief flapping of their wings. It showed that SDS chickens exhibit a sudden attack prior to death lasting an average of 53 s and characterized by failure of balance, violent flapping and strong muscular contractions [2].

The underlying physiological mechanisms that result in SDS are unknown, but are thought to be diet-related metabolic disturbances leading to cardiac

dysfunctions [3]. This is likely associated with the genetic selection of broilers for growth and feed conversion efficiency, while neglecting basic physiological requirements [4]. It has been recognized as a specific condition since the 1950s, when broiler chickens were begun to be grown commercially in large numbers [5] and leads to economic losses in the broiler industry worldwide every year. Subsequent high mortality rates and inherent financial losses are tremendous and they keep on increase each year [6]. Young, healthy, fast-growing boiler chickens die suddenly while standing, walking, sparring, or feeding. They die with a short terminal wing-beating convulsion and are frequently found on their back [5]. From a biochemical point of view, data for SDS is limited because of the lack of consistent behavioural symptoms that allow SDS identification before death [2, 7].

Many nutritional, environmental, genetic and management factors have been suggested to cause SDS. The role of nutritional factors and diet manipulations is very important to achieve genetic potential and prevention of metabolic disorders.

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Although the physiological and pathological aspects of metabolic and cardiovascular diseases in broilers have been investigated in detail in previous studies, the role of nutrition in the aetiology of metabolic disorders, especially SDS has been limited. Therefore, in this overview, various aspects of nutrition on SDS were evaluated.

Occurrence and Incidence: The exact time of SDS onset is different and usually a range was account for starting and incidence of SDS in broiler. It is reported that all ages of broilers are affected, starting as early as 2 or 3 days of age [2, 8, 9] and ongoing through to market age [9]. Peak mortality usually occurs in broilers between 21 and 28 days of age [10]. Males are more often affected than the females and account for 60-80% of the deaths [11]. A higher growth rate and hormonal involvement may be responsible for the high susceptibility of the males. The percent mortality of 1.31-2.46 [12], 0.90-3.61 [13], 0.71-4.07 [14] and 0.5-5.0 % [7, 15] were reported due to SDS in broilers. In healthy flocks, it is the most frequent cause of death and accounts for up to 4% mortality in some all-male flocks [8, 16]. Therefore time of SDS onset could be multifactorial.

Fast growing broilers have a high incidence of SDS [1] and it consider as metabolic stress. Thus, the susceptibility of broiler chicken to SDS is co-related with metabolic characteristics imposed by rapid growth. The faster-growing males make up approximately two-thirds of the affected birds [2, 8]. The incidence of SDS could markedly decreases when the growth rate in broilers is controlled by means of growth rate control methods (genetic selection, dietary restriction, feed deliver, dietary composition and form, light programs, etc.).

The peak of SDS in chickens is at approximately 2-4 wk of age [7, 15]. These losses are costly to the broiler industry. Thus, as stated above, SDS is very important to the economic profit from 2 aspects. First, high percentage of mortality and second, bird's death in old age that leads to increase of expenditure of maintenance.

Aetiology: The death in broilers succumbing to SDS is associated with various factors. There are numerous genetic [17] management (lighting [18-20]; stocking density [18, 21, 22]; exercise [9, 18]; social interaction [23] and noise) nutritional and physiological factors that attribute to SDS. SDS is frequently, but not exclusively, accompanied by some failure of the cardiovascular system. The aetiology of SDS in broilers is poorly understood, but fast-growing broilers have a high

incidence [1]; thus, any problems that arise from pressure of high metabolic rate because of selection for rapid growth and better feed conversion efficiency can account as a cause of SDS.

In the rest of matter nutritional aspect of SDS will consider hope that can be made correct conclusions in this regards.

Diet Texture: The form of diet is very important to the rate and time spent on feed intake. Feed intake itself influences the metabolic rate and finally metabolic diseases. It is indicated that the incidence of SDS was significantly reduced when the birds were fed by mash rather than pelleted diets [13]. Unfortunately, the result was screwy by reduction in the growth rate. Due to pelleted feeds induced more rate of feed intake and faster growth rate hence incidence of SDS are more in broilers [24]. Others [25-27] have reported that broilers will grow slower when fed mash as compared to crumbled/pelleted diets. The incidence of SDS can reduce after slower growth. Thus, we can't directly connect feed form (mash, pellet, crumble) to incidence of SDS and this reduction is doubtful.

In another experiment it suggested that, when the feed was pelleted, some changes occurred in the diets affect SDS and is independent of body weight [28]. They proposed that toxic agents were produced when protein supplements were subjected to pelleting and this induced SDS. They stated that reduction in the quantity of soybean meal in the diets reduced the incidence of SDS; therefore, toxins could be produced when the soybean meal is pelleted. It is more likely that the inclusion of meat meal sources have unknown factor(s) which provides some protection against the incidence of SDS, but this hypothesis remained non-specific.

Diet Composition

Energy Sources [: It is believed that energy sources can influence the incidences of SDS. The replacement of carbohydrate with lipids was investigated. The different percent mortality was observed by use of glucose (6%) lipids (2.1%) and corn starch (2.5%) as main sources of energy in diets [29]. These results are probably related to lactic acid metabolism. It is shown that various levels of tallow in diets had a slight effect on the incidence of SDS [30]. Dietary intervention has also been used to limit growth; although, Classen [20] considered the use of low-energy diets to have a relatively minor capacity in controlling growth and probably incidence of SDS, but Scott [31] showed that, as compared to high-density diets, low-density diets significantly reduced SDS mortality.

Moreover, other reported that the incidence of SDS was higher in flocks supplied by a feed company that use less corn and more wheat than other feed companies [14]. Therefore, it appears that the incidence of SDS is higher in broiler chicks fed with wheat-soybean meal than in those fed with corn-soybean meal based. However, it is appear that obtained lower body weight by various energy sources led to reduce incidence of SDS not and energy sources led to lower incidence of SDS.

Protein: It seems that dietary protein solely has little influence on SDS. The use of protein-deficient diets will reduce growth and likely SDS, but generally it increases the deposition of carcass fat [20] which increases the cost of production. Significantly lower incidence of SDS (50%) in 29-56 day old broiler chickens fed with 24% protein finisher diet related to birds fed a 19% finisher diets was recorded [32]. However, the age of SDS occurrence is considerable in this regard. Moreover, those diets consist of different ingredients which difficult explanation of results. If high protein diet containing excess level of sulphur is given to the birds, increased acid secretion results in higher level of lactic acid causing the condition SDS. In another study, the incidence of SDS was reported to decrease when meat meal was used in the diet [33]. They concluded that meat meal sources provided unknown factor(s) which prevented birds against SDS. Unfortunately, other factors exist in the study of Blair *et al.* [33] that affect the results. For example, level of diet phosphor in the basal diet (soybean based) which was higher, but in the test diet (meat meal based) supplemental phosphorus was not done. Similarly, diets including meat meal have lower salts and supplemental fat. These slight changes in the diet ingredients can have considerable effects on the incidence of SDS. Such examples are problems that nutritionists face when investigating the nutritional factors involved in the incidence of SDS.

Amino Acids: Relatively few studies were done regarding the role of amino acids (AAs) on SDS. Likely protein, graded increase of AA levels can influence growth rate and indirectly affect the incidence of SDS. The taurine metabolism had more attention between AAs in this regard. Taurine is an unessential AA for poultry and found in a majority of animal tissues. The uppermost accumulation of taurine is in the heart tissue and considered as 50% of body free taurine reserves. Poultry species produce enough amounts of taurine; however, reduction in taurine levels can lead to heart failure, which may induce the SDS condition.

In relation to the role of meat meal in SDS, shown that taurine had no major position in aetiology of SDS [34]. However, taurine affects calcium metabolism in the heart [35] and can occupy a position in the SDS. However, it demonstrated that taurine role in the SDS is very colourless [36].

Lipids (Prostaglandin): The level and type (saturated or unsaturated) of fat in the diet may be involved in SDS. Rotter *et al.* [37] have shown that the type of lipid influences the incidence of SDS and birds fed with saturated oils had the lowest occurrence of SDS. Furthermore, it illustrated that broilers fed with sunflower oil had lower mortality from SDS than broilers fed with tallow [38]. These authors concluded that lipid supply and, probably, the saturation rate is related to SDS.

Supplemental dietary lipid is typically provided as animal tallow or hydrogenated oils. These sources of lipids are rich in saturated (trans, n-6) and poor in unsaturated (n-3) fatty acids. The precursors of long chain n-6 and n-3 fatty acids (arachidonic and eicosapentaenoic acids [EPA]) are essential fatty acids of linoleic (18:2 n-6) and linolenic (18:3 n-3) acids, respectively. Phospholipase A₂ mobilizes the ester-linked arachidonic acid and EPA and produces free arachidonic acid and EPA, which can act as substrates for cyclooxygenase and lipooxygenase to produce eicosanoids. An imbalanced condition of eicosanoid synthesis may induce cardiac diseases and sudden death in fast-growing broiler birds.

After hatching of the chick, the bulk of the lipids in the myocardial tissues that originated from the yolk lipids are incorporated into the phospholipids of the structural components. Variations in the tissue phospholipids caused by maternal diet can alter eicosanoid synthesis in the cardiac tissue. Thus, developing dietary strategies for enhancing tissue n-3 fatty acid content in broiler chickens without affecting growth and product quality is critical. The effect of yolk fatty acids was investigated on the long-chain n-3 fatty acid content of tissues (heart) and eicosanoid production [39]. It was concluded that adjusting maternal dietary n-3 fatty acids and following the yolk fatty acids enhances tissue retention of n-3 fatty acids through growth and reduces eicosanoid production, which could lead to fewer metabolic-related disorders (SDS) in poultry. In another experiment, feeding breeder hens to obtain eggs with low, medium and high levels of n-3 polyunsaturated fatty acids (PUFA) were investigated on SDS incidence [40]. It appears that the maternal reserves (yolk) have the integral involvement in the

retention of n-3 long-chain PUFA in the cardiac tissue of offsprings. Dietary imbalance and the underlying requirement regarding n-3 fatty acids for tissue incorporation during growth ultimately caused the conditions that predispose broilers to SDS. On the other hand, higher rate of free radical formation occurs in broiler chickens as a consequence of high intensity of metabolism. Free radicals may play an important role in the aetiology of metabolic diseases like SDS [3]. Addition of 'melatonin' to the diet of broiler chicken tends to decrease the incidence of SDS. As shown above, the incidence of SDS increases under high-intensity light [18], which has an inhibitory effect on melatonin production [41].

In SDS birds, linoleic acid and arachidonic acid of heart tissue are low; hence, decrease in the synthesis of prostaglandin leads to deterioration of membrane structure of the heart and cardiovascular disorders, heart dysfunction and rise in the incidence of SDS [24].

Carbohydrates: There is some evidence that carbohydrate availability and type, rather than protein or fat, are part of the pathogenesis [16]. The different percent and delay mortality were observed when glucose and corn starch were used as main sources of carbohydrate in diets [42]. These results probably are linked to lactic acid metabolism and it must be noted that changes in the lactic acid levels is premier rather than the absolute amount of lactic acid.

Lactate: The serum concentration of lactate dehydrogenase (LDH), glutamic oxaloacetic transaminase and creatine phosphokinase are elevated in SDS, which are used as indicators for clinical diagnosis of circulatory disturbance in humans [7]. This issue shows that the amount of substrate of these enzymes is elevated and likely be related to SDS occurrence. The cardiac system readily damage by an elevation in the serum concentration of lactic acid, a metabolite of glycolysis. It is shown that intravenous lactate injection causes an elevation of lactic acid concentrations in blood and SDS-like death in broilers [43]. On the other hand, lactate accumulation in blood and muscles can lead to pain, failure of balance, strong muscular contractions, ultimately violent flapping and overturning. Furthermore, inadequate oxygen supply because of insufficient growth of visceral organs (heart, lung, etc.) results in hypoxia and lack of aerobic metabolism. These reactions result in the production of lactate from pyruvate by LDH and increased lactic acid production leads to change in blood pH, acidosis, cardiovascular system damage and cardiac arrhythmia.

In addition, because of more feed intake, more lactic acid is produced in the intestines (digestion or fermentation) and may absorb more, influence the acid-base balance and SDS incidence

Electrolytes (Acid-Base Balance): The fact that death is apparently due to heart failure may suggest the involvement of electrolytes (Na^+ , K^+ , Cl^-) in SDS. The serum levels of various electrolytes, including Na^+ and K^+ was compared from SDS broilers and matched healthy broilers [12]. They reported that the values of SDS broilers were not consistently different from those of healthy birds. Signs similar to those seen in the SDS condition are observed after injection of electrolytes such as potassium in the heart or after force feeding lactate, which confirm acid-base imbalance. Acid-base imbalance likely is a reason. If electrolyte imbalance is the cause of other predispose agents is not clear and further investigations are essential in this case.

Minerals: It is proposed that stress condition (lighting and stocking density) increase catecholamine secretion from adrenal gland and increase Ca^{++} levels in the cardiac muscles. This event can lead to cardiac arrhythmia [9]. On the other hand, to prevent leg problems in fast-growing broilers, broiler diets are commonly supplemented with vitamin D_3 above the recommended levels. Therefore, there is a risk of over supplementation. It has been established that high supplementation of vitamin D_3 causes a variety of metabolic and pathological lesions in the cardiomyocytes [44]. It is pointed out that over-supplementation of vitamin D increases the risk of SDS in broilers [45].

Additionally, selenium deficiency has been considered as causing aetiological agents of SDS in broilers. The role of selenium may be related to the antioxidant role of selenium against free radical. Alternatively, it suggested that SDS is similar to tetany (Mg deficiency), which is induced by mineral interactions [8]. For example, when high levels of saturated fatty acids are used in diet, they form complexes with minerals (Mg, Ca and Zn) and reduce mineral bioavailability. This reaction can influence the nervous system and cause a SDS-like condition. Nonetheless, it illustrated that Ca, P and Mg supplementation (0.2%) has no effect on SDS [8].

Vitamins: The presence of a blood clot in the heart may suggest that the level or absence of certain vitamins in the diet might be involved in SDS. There is little evidence that vitamins have an effect on the incidence of SDS in

commercial broilers [5]. It is reported a higher incidence of SDS in flocks supplied with wheat than in those fed with other feed [14]. The possibility exists that B vitamins (Biotin) due to deficiency in the wheat was responsible of this higher occurrence. The various roles of B vitamins in body metabolism can involved in metabolic disorder (SDS) and must be noted. Additional biotin [46] provided by corn or wheat may have contributed to the decreased mortality found with corn diets although the biotin requirement should have been met by the amount of premix added to each diet. However, addition of biotin (up to 100 µl/d) had no advantages in SDS condition [47]. Nutritionists always face with simple dietary deficiency of vitamins or deficiencies induced by nutrient interactions. Thus, such problems harden the responsibility and recognition of nutritionists.

Drugs: The heart function is related to SDS. Therefore, some drugs have been examined to SDS, but have generally not been successful. It confirmed that 'aspirin' had no beneficial effects on the SDS condition [13].

Anti-coccidiosis drugs, especially ionospheres anti coccidiosis, appear to have important role in the SDS. It is showed that, in birds fed with ionospheres, the incidence of SDS increased considerably [42]. Moreover, it is revealed that the incidence of SDS was higher in flocks fed with monensin (ionosphere) in both starter and finisher diets [14]. Ionosphere components affect the transmission of ions across cell membranes. Sodium and potassium are the main ions influenced by ionospheres. In addition to movement of ions, permeability of the membrane related to hydrogen ion increase and acid-base balance changed. Acid-base imbalance can lead to degeneration of the heart muscles and problems regarding SDS condition. Interference of ionosphere in the incidence of SDS not clearly understands and up to now ionosphere toxicity has not been reported. On the other hand, ionospheres alter the transmission of ions and these events are associated with normal metabolic functions like the events that arise in SDS, but we can not found reliable relevance in this case.

Diagnosis: There are no diagnostic lesions in broilers that have died from SDS, but suddenly death in a previously healthy broiler [11, 48]. There is a specific association with feed intake. The condition was first reported as being caused by lung oedema [49] and the lungs are oedematous, except in the freshly dead birds [21]. The bursa is large and normal, which again suggests that the bird was healthy immediately prior to death. Heart

ventricles are contracted and the atria are dilated and filled with blood, a significant observation. There are no specific gross or histopathological lesions that would distinguish broilers that have died from SDS from the healthy broilers that had been euthanized [5].

CONCLUSION AND RECOMMENDATION

The judgment concerning risk factors for SDS is difficult, but have been suggested a variety of nutritional and environmental factors. The chain of events that occurs and leads to SDS in broilers is complicated and the role of nutrition in reducing SDS in broiler warrants further investigations. Hence, it appears that the role of nutrition in comparison to other factors, especially genetics, is very low and problems occurred by incorrect selections can not prevent by nutritional strategies. As discussed above one can list long nutritional recommendations to reduce or prevent SDS incidence (a) Lowering energy intake by changing feed texture, density, restriction b) The use of proper amounts of vitamins, antioxidant, saturated lipids, essential fatty acid c) The provide of proper acid-base imbalance, Ca, Mg, P, K, Se content of diet), but it must take into account that effects of each are not definitive and obvious. Stress (genetic, nutritional, environmental and management) is the main factor to contribute towards the pathogenesis of SDS. Posterior stress conditions, optimal and normal physiological state were abrupt. Hormonal and body secretion were changed. Normal permeability of capillaries and membranes were altered. Blood pH and pressure were disturbance. The circulatory system was affected. Cardiac arrhythmia and heart dysfunctions may occur and finally can take a place SDS.

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