THE INFLUENCE OF DIET COMPOSITION AND TISSUE TAURINE CONTENT ON THE INCIDENCE OF SUDDEN DEATH SYNDROME IN MALE BROILER CHICKENS

by

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ABSTRACT

Five experiments were conducted with male broiler chickens to study the influence of diet composition on the incidence of Sudden Death Syndrome (SDS).

In Experiment 1, the effects of 4 levels of dietary lactate (0, 2.5, 5.0 and 7.5% calcium lactate) and 4 levels of dietary glucose (0, 15, 30 and 45% cerelose) were studied in a factorial experiment with 1280 chicks reared in battery brooder cages to 4 weeks of age. There were no significant differences in either total mortality or mortality due to SDS.

In Experiments 2, 3 and 4, the chicks were fed isocaloric and isonitrogenous diets with either corn or wheat as the grain type and meat meal or soybean meal as the main protein source. In Experiments 2 and 3, the chicks (6000 and 9600, respectively) were raised in floor pens to 6 weeks of age while in Experiment 4, the chicks (640) were reared in battery brooder cages to 4 weeks of age. SDS mortality was affected by both cereal type and protein source. Broilers fed wheat based diets had a higher incidence of SDS mortality than those fed corn based diets. The incidence of SDS was higher when meat meal was excluded from the diet.

In Experiment 5, the chicks were fed diets supplemented with guanidinoethyl sulfonate (GES), a taurine transport inhibitor in rats. At 4 weeks of age broilers receiving 1.50% dietary GES had significantly lower (p<.05) cardiac taurine concentrations, but significantly higher (p<.05) brain taurine concentrations than controls. There was no effect of GES supplementation on total mortality or the incidence of SDS.
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THE INFLUENCE OF DIET COMPOSITION AND TISSUE TAURINE CONTENT
ON THE INCIDENCE OF SUDDEN DEATH SYNDROME
IN MALE BROILER CHICKENS

1.0 INTRODUCTION:

Sudden Death Syndrome (SDS) refers to the condition in which apparently healthy, fast-growing broiler chicks die suddenly from no apparent cause. There is usually a short, wing-beating convulsion prior to death so that the majority of affected broilers are found dead lying on their backs. As a result, the condition is often referred to as "Flip-Over disease". SDS has also been referred to as "Acute Death Syndrome", "heart attack" and "fatal syncope" (Merck, 1986).

Broilers of all ages are affected starting as early as two days of age and continuing through to market age (6 1/2 weeks for broilers and 9 weeks for roasters). Peak mortality usually occurs between 3 and 4 weeks of age (Brigden and Riddell, 1975; Ononiwu et al., 1979b; Gardiner et al., 1988b). In Ontario, however, the period of peak mortality appears to be occurring at a younger age and in 1986 was reported to be between 8 and 20 days of age (Bowes and Julian, 1986).

With most of the major disease problems under control, the economic importance of SDS has become more apparent. Since death occurs principally from 3 weeks of age on, the economic loss incurred involves not only the initial cost of the chick, but also the cost of the feed consumed prior to the chicken's death.

SDS is recognized as a major cause of mortality in broiler flocks throughout Canada. Cassidy et al. (1975) reported
incidences of 2.16% in experimental broiler flocks at the Agriculture Canada Research Station in Kentville, Nova Scotia in Eastern Canada. The average incidence in Western Canada that same year appeared to be somewhat lower. Brigden and Riddell (1975) reported an average incidence of 1.13% in 4 broiler flocks in Alberta and Saskatchewan. By 1985 the average level of SDS in Alberta and Saskatchewan appeared to have increased. In a survey of 51 broiler flocks, the average incidence of SDS was 1.95% (Riddell and Springer, 1985). Levels in individual flocks, however, varied from .71 to 4.07%. In Ontario, SDS was reported to affect 1.05% of the broilers housed, representing more than 30% of total mortality (Bowes and Julian, 1986).

The SDS reported in Canada appears to be the same as the conditions referred to as "lung edema" in England (Hemsley, 1965) and "died in good condition" in Australia (Jackson et al., 1972).

The incidence of SDS reported in Canadian broiler flocks is somewhat higher than those reported for flocks in England and Australia. Hemsley (1965) reported an average incidence of .46% in 14 broiler flocks in England. This represented 23% of total mortality. The survey was done at least 10 years prior to those done in Canada. It is possible the levels of SDS in England have increased to the levels recently reported in Canada. Jackson et al. (1972) surveyed nine flocks on four broiler farms in New South Wales, Australia and an average of .65% of broilers housed (or 15.6% of total mortality) were classified as having "died in good condition". This included SDS affected broilers as well as broilers that died from known panics. When only SDS
affected broilers are considered, the average is reduced to .46% of broilers housed (or 11% of total mortality). In 1982, however, Steele and Edgar, also from Australia, reported an average incidence of 2.4%, or 36% of total mortality. This was, however, in a single broiler flock and in Western Australia rather than in New South Wales.

SDS is difficult to study because of its relatively low rate of occurrence and because no particular behavioral or environmental event has been identified as preceding death (Newberry et al., 1987). Various dietary and management practices, as well as a variety of chemicals, have been studied to try and alter the occurrence, but the results have been inconsistent from study to study.

The objective of this series of experiments was to find a dietary regime which would increase the incidence of SDS so that its cause could more easily be studied. The following hypotheses were tested:

1. Supplementation with lactate in the diet will induce SDS.
2. Glucose fed broilers are more susceptible to SDS than corn starch fed broilers.
3. The incidence of SDS is higher for wheat fed than corn fed broilers.
4. Exclusion of meat meal from the diet of male broilers will increase the incidence of SDS.
5. Supplementation with guanidinoethyl sulfonate, a taurine transport inhibitor, will reduce tissue taurine levels and induce SDS.
2.0 LITERATURE REVIEW - SUDDEN DEATH SYNDROME:

2.1 Cause of death: The actual cause of death in SDS cases is unclear and no specific diagnostic lesions are associated with the syndrome. Well fleshed, otherwise healthy broilers, found dead on their backs are usually assumed to have died of SDS since that position is rare in death from other causes (Merck, 1986). SDS affected broilers cannot, however, be diagnosed solely on the basis of their being found on their backs. Newberry et al. (1985) noted that some SDS affected broilers were found dead lying on their sternums while other broilers not diagnosed as having died of SDS were observed to have a sudden attack and were found dead lying on their backs. Affected broilers are well fleshed and were eating normally as indicated by a full or partially full crop containing normal ingesta, feed in the gizzard, distended intestines filled with semi-solid digesta and mucus, and a small or empty gall bladder. The ventricles of the heart are usually contracted and the auricles dilated and filled with blood.

No particular bacterial or viral agent has been identified in broilers that died from SDS (Jackson et al., 1972; Bridgen and Riddell, 1975). Some researchers have found lung congestion and edema and have proposed that the lung congestion was a result of heart failure and that the broilers suffocated (Brigden and Riddell, 1975; Ononiwu et al., 1979a). Further studies have shown that lung congestion is not a consistent feature and that freshly dead broilers diagnosed as dying from SDS had no lung congestion (Riddell and Orr, 1980). This suggests that lung congestion is a postmortem change probably
related to position at death.

Cassidy et al. (1975) observed "blood structures" in the heart chambers of broilers dying from SDS. The structures were found in all four heart chambers with gross appearance unaffected by age or sex. They had a shiny, smooth appearance and were molded to the shape of the chamber. Since these structures are common in broilers affected by SDS, it was thought that they were the cause of death. Histological examination of these structures, however, showed that they were composed of erythrocytes, leucocytes, fibrin and serum with no fibroblasts, collagen fibers or thrombocyte agglutination present. This composition would indicate that the structures were postmortem blood clots.

SDS death appears to involve heart damage. Microscopic examination of cardiac muscle from SDS affected broilers showed degeneration of the fibers, separation of the fibers by edema, and infiltration of heterophils (Ononiwu et al., 1979a). Julian and Bowes (1987) have suggested that death is due to left ventricular fibrillation, but there is still no conclusive evidence as to the cause of this fibrillation.

2.2 Predisposing factors: Several factors have been investigated for possible involvement in the etiology of SDS. In many cases the results have been inconsistent from study to study.

2.2.1 Sex: The incidence of SDS is clearly influenced by the sex of the broiler since more than 70% of SDS affected broilers are male (Hemsley, 1965; Brigden and Riddell, 1975; Steele and Edgar, 1982; Riddell and Springer, 1985). It is not
clear whether this is due to their faster growth rate or to some endocrinological effect on metabolism.

Gardiner et al. (1988a) injected estradiol-17β-monopalmitate into male broiler chicks to try and alter the incidence of SDS. The estradiol treatment inhibited early male sexual development so that at 9 weeks of age combs and testicles from the treated broilers were 1/4 the weight of those from the controls. There was no effect of the estradiol treatment on the incidence of SDS.

2.2.2 Genetics: Hemsley (1965) observed that SDS mortality was lower with broilers of slower growing breeds, such as the Light Sussex crosses, than with broilers of three White Rock strains (Arbor Acre, Cobb and Pilch). When females of four different maternal breed types were mated to cornish males, the progeny of the "Cobb" female had twice the incidence of SDS as the progeny of any of the other breeds. Newberry et al. (1985) noted differences in the incidence of SDS between broilers of two strains used in a study on the effect of alternating light. It would appear that there may be a genetic basis for the incidence of SDS. Chambers (unpublished data), however, stated that the heritability of SDS was low and concluded that non-genetic methods should be considered for reducing SDS mortality.

2.2.3 Growth rate: The influence of sex and genetics on the incidence of SDS has often been attributed to differences in growth rate (Cassidy et al., 1975). Ononuwi et al. (1979b) observed that broilers dying from SDS were the heavier broilers in the flock. In addition, Gardiner et al. (1988b) demonstrated that the incidence of SDS increased with body weight of the
flock. Brigden and Riddell (1975), however, indicated that SDS affected broilers were of average weight.

Mollison et al. (1984) reported that feed restriction, and thus slower growth rate, did not decrease the incidence of SDS. A 13% feed restriction was used and a 15% reduction in mean body weight obtained. Bowes et al. (1988), using a 25% feed restriction, reported a 41% reduction in mean body weight and obtained no SDS mortality out of the 300 broilers housed. Although the number of broilers used in the study was low, this would appear to support the hypothesis of relationship between growth rate and the incidence of SDS. A feed restriction of greater than 13%, or a mean body weight reduction of greater than 15%, would appear to be required to reduce the incidence of SDS.

Newberry et al. (1985) noted that one strain of male broiler chickens had a significantly lower SDS mortality but a higher mean body weight than a second strain used in the same trial. This would suggest that genetics, and not growth rate, is a major predisposing factor in the etiology of SDS. It also suggests that it is possible to use genetic manipulation to reduce the incidence of SDS without depressing growth rate.

2.2.4 Time of year: Gardiner et al. (1988b) observed seasonal fluctuations in the incidence of SDS in male broiler flocks. Those broiler flocks started in the winter months had a higher incidence of SDS than those started in the summer. No reasons for this relationship were proposed.

2.2.5 Stress: Environmental stressors such as noise, social interaction, stocking density and sudden operation of
feed auger motors, have been implicated in the etiology of SDS (Ononiwu et al., 1979b; Steele and Edgar, 1982). Using continuous video tape recordings of individually marked male broiler chickens, Newberry et al. (1987) demonstrated that no particular behavioral or environmental event occurred prior to death by SDS. None of the SDS affected broilers died following a sudden loud noise, agonistic interaction, cannibalism or piling up of broilers. Rotter et al. (1985) showed that stocking density (.09 vs .08 m$^2$/broiler or 11.1 vs 12.5 broilers/m$^2$) had no effect on SDS mortality. It is therefore unlikely that stress plays a major role in the etiology of SDS.

Gardiner and Hunt (1984) studied the possibility of reducing the incidence of SDS by inclusion of the compound reserpine (3,4,5-trimethoxy-benzoyl methyl reserpate) at low levels (0 to 3 mg/kg) in the diet. Resperine is an anti-hypertensive and tranquilizing agent that has been included in poultry diets as an anti-stress compound. They concluded that neither total mortality nor mortality due to SDS was affected by dietary reserpine.

2.2.6 Lighting: Ononiwu et al. (1979b) reported that the incidence of SDS in mixed-sex flocks could be reduced by using intermittent (1 h light: 3 h dark per 4 hour period phased in over a 14 day period after 10 days of age) rather than continuous lighting. Total mortality was not affected indicating that mortality due to causes other than SDS increased. Carcass fatness was reduced and there was improved feed efficiency. The effect of intermittent lighting on mean body weight was not reported. Cave (1981) indicated that mortality, including SDS,
was not significantly different between broilers of both sexes receiving intermittent light (1 h light: 3 h dark per 4 hour period from day old) and those receiving continuous light. Again carcass fatness was reduced and there was improved feed efficiency. There was no effect of lighting program on weight gain. Newberry et al. (1985) studied the effects of rearing male broilers to roaster weight under a program of alternating bright light from side-to-side within pens at regular intervals, versus continuous lighting. The light treatment had no effect on SDS mortality, mean 70-day body weight or feed to gain ratios.

Light intensity has been shown to affect broiler performance. Skoglund and Palmer (1962) indicated that broilers were heavier when reared under intensities of 22 or 54 lux than when reared under very high intensity (1292 lux) light. Ononiuwu et al. (1979b) proposed that if light intensity was above optimum it would induce cannibalism, excitement, fighting and piling. Such activities would add to the stress of the broilers. Newberry et al. (1985) observed that broilers were more likely to be affected by SDS in the lighted areas (6 or 12 lux) than in the darker areas of the pen (.5 lux). Light intensity was, therefore, investigated for possible effect on the occurrence of SDS. Riddell and Springer (1985) failed to find a correlation between mortality due to SDS and light intensity within the range of .1 to 13.2 lux. Newberry et al. (1986), using light intensities varying from .1 to 100 lux, reported that light intensity had no effect on total mortality or mortality due to SDS. It is therefore unlikely that light intensity is a
2.2.7 Nutrition: Several dietary factors have been investigated for their involvement in the incidence of SDS including levels of minerals, vitamins, protein, energy and fat.

2.2.7.1 Minerals: A condition similar to SDS in broilers has been identified in broiler breeders and commercial hens at onset of egg production (Hopkinson et al., 1983; Pass, 1983). The condition was shown to be due to a dietary potassium deficiency. Supplementation with potassium bicarbonate in the water (.62 g/layer) or potassium carbonate in the feed (.36%) prevented the syndrome. Hunt and Gardiner (1982) demonstrated that supplementation with .1, .2 or .3% potassium carbonate to a wheat-soy diet did not influence total mortality or the incidence of SDS in male broiler chickens.

Dietary fat forms calcium and magnesium soaps in the digestive tract of chickens making these minerals unavailable for absorption. Julian (1986) proposed that SDS was caused by acute hypomagnesemic tetany as a result of magnesium lost in the faeces. Supplementation with .2% calcium, .2% phosphorus and .2% magnesium, however, had no effect on the incidence of SDS.

Bowes et al. (1989) compared the serum biochemical profiles of male broilers with female broilers and male White Leghorn chickens. (SDS has not been identified in White Leghorn chickens). There were no significant differences in serum potassium, phosphorus or magnesium levels. Serum calcium levels in male White Leghorns were, however, found to be significantly higher than those found in male broilers at 9 days of age. There were no significant differences at 20, 30 or 42 days of
age. Riddell and Orr (1980) compared the potassium, magnesium, sodium and glucose levels of blood and hearts from broilers dying from SDS with those of normal healthy broilers. They observed no consistent differences.

Rotter et al. (1985) reported that the calcium in the heart tissue of SDS broilers was significantly higher than in culled broilers. In subsequent studies, however, Rotter et al. (1987) and Rotter et al. (1988) observed no significant differences in cardiac calcium levels between these two groups.

2.2.7.2 Vitamins: Hulan et al. (1980) studied the effects of biotin, pyridoxine and thiamin supplementation, singly or in combination, on the incidence of SDS. The B vitamins function principally as coenzymes. For example, biotin is involved in carboxylation reactions, pyridoxine in protein metabolism and thiamin in dekarboxylation of α-keto acids and in the transketolation reaction in the monophosphate shunt pathway of glucose metabolism. Inclusion of biotin alone, at 300 μg/kg diet (2 times NRC requirements), significantly reduced total mortality (p<.005) and the incidence of SDS (p<.05). Inclusion of biotin at the same level, but combined with 5 mg pyridoxine per kg diet (1.7 times NRC requirement) and 3 mg thiamin per kg diet (1.7 times NRC requirement) did not significantly affect SDS mortality. No reasons for this interaction were proposed. Steele et al. (1982) reported that the occurrence of SDS was unaffected by supplementation of high levels (1.2 to 25 times NRC requirements for different stages of growth) of biotin in the water. Uptake was confirmed by radioisotopic analysis of liver biotin. Whitehead and Randall (1982), Hunt and Gardiner (1982), and
Mollison et al. (1984) also failed to find any effect of biotin supplementation on the incidence of SDS. It is therefore unlikely that dietary biotin is involved in the etiology of SDS.

2.2.7.3 Protein: The effect of dietary protein level on the incidence of SDS is unclear. Mollison et al. (1984) observed that the incidence of SDS was significantly reduced by feeding a 24% protein finisher diet as compared to feeding a 19% finisher diet. The diets were isocaloric (3100 kcal AME/kg). Julian and Bowes (1987), however, reported no significant differences in the incidence of SDS among broilers fed isocaloric (3100 kcal AME/kg) diets with high (31%), medium (24%) or low (17%) protein content.

2.2.7.4 Energy: Julian and Bowes (1987) studied three dietary energy sources and their effect on the incidence of SDS. In flocks fed diets with similar energy (3100 kcal AME/kg) and protein (24%) content, the occurrence of SDS was higher when glucose monohydrate was the energy source as compared to tallow or corn. They hypothesized that SDS is related to a problem with carbohydrate metabolism. There may be a protein x cereal interaction affecting the incidence of SDS. Julian and Bowes (1987) reported that the incidence of SDS was significantly higher in broilers reared on a high protein (28%), high caloric (3100 kcal AME/kg) diet than those reared on a low protein (18%), low caloric (2400 kcal AME/kg) diet.

2.2.7.5 Fat: Rotter et al. (1985) proposed that fat metabolism may be involved in the etiology of SDS and that dietary fat type may play a role in prevention of its
occurrence. They reported that the incidence of SDS, but not total mortality, was lower when a wheat-soy diet was supplemented with sunflower oil (an unsaturated fat) than when the same diet was supplemented with tallow (a saturated fat). In a later study (Rotter et al., 1988), the effects of hydrogenated coconut oil (HCO), tallow, sunflower oil (SFO) and tallow/SFO on the incidence of SDS were investigated. There was no effect of dietary fat type on overall mortality. SDS mortality between 0 and 4 weeks of age, however, was significantly lower for the broilers receiving the HCO (a saturated fat) diet as compared to those receiving the SFO (an unsaturated fat) diet. This contradicts the earlier findings that feeding an unsaturated fat resulted in a lower incidence of SDS than feeding a saturated fat.

2.2.7.6 Fatty Acids: Rotter et al. (1985) analyzed the fatty acid composition of heart and liver tissues from SDS affected broilers in comparison with those from culled broilers. In the heart, palmitic (16:0) and oleic (18:1) acids were higher in the tissues from the SDS affected broilers while the linoleic (18:2) and arachidonic (20:4) acids were lower. In the liver, oleic acid levels were higher in SDS affected broilers while linoleic and arachidonic acids were lower. Linoleic acid is converted to arachidonic acid which in turn is a precursor of prostaglandins. Prostaglandins have a wide variety of roles including modulating and regulating myocardial contractile force, heart rate, and cardiac rhythms. Rotter et al. (1985) proposed that a deficiency of arachidonic acid would reduce the amount of prostaglandins that would be synthesized so that heart
function would be disrupted leading to fibrillation or arrhythmia. Since biotin plays a role in the conversion of linoleic acid to arachidonic acid, Rotter et al. (1985) also hypothesized on the role of biotin status in the etiology of SDS. In a subsequent study, Rotter et al. (1987) failed to find an effect of dietary linoleic acid concentration on the incidence of SDS. Therefore, the role of linoleic acid in the etiology of SDS remains questionable.

As previously stated, Rotter et al. (1985) reported that cardiac and hepatic arachidonic acid levels were lower in SDS affected broilers than in culls. In a subsequent study, however, Rotter et al. (1988) compared the fatty acid composition of heart and liver tissues from SDS affected and normal, healthy broilers. They observed significantly reduced arachidonic levels in hearts from the SDS broilers but no differences in liver levels. Wu and Nakue (1987), however, reported decreased arachidonic acid levels in the liver of SDS affected broilers when compared to levels in controls. Contrary to the results already presented, Buckley et al. (1987) reported that heart and liver tissue arachidonic acid levels were generally higher in SDS than in control chickens.

Buckley et al. (1987) observed significantly higher levels of unsaturated fatty acids, as indicated by the double bond index:saturated fatty acid ratio, in the hepatic triacylglycerol fraction of SDS broilers and hypothesized that this may increase membrane fluidity disrupting the activity of membrane bound enzymes. A reduction in prostaglandin synthesis cannot, therefore, be ruled out as a cause of SDS.
2.2.7.7 Cereal Type: The involvement of cereal type on the etiology of SDS is unclear. Hunt and Gardiner (1982) stated that there were no differences in total mortality or mortality attributed to SDS between broilers fed wheat-soy or corn-soy based diets. On the other hand, Mollison (1983) reported that broilers fed wheat-soy diets had higher incidences of SDS than those fed corn-soy diets. In a later study, however, Mollison et al. (1984) showed that corn-fed broilers had significantly better weight gains and feed to gain ratios than wheat-fed groups, but total and SDS mortality were not affected. In a survey of mortality in 51 broiler flocks in Alberta and Saskatchewan, Riddell and Springer (1985) noted a relationship between the incidence of SDS and the type of dietary cereal used. The incidence of SDS was higher in flocks supplied by a feed company which used less corn, and more wheat, than the other feed companies used by the broiler farms surveyed.

2.2.8 Pelleting: Proudfoot and Hulan (1982) and Hulan and Proudfoot (1987) observed higher SDS mortality in male broiler chickens fed a crumbled-pellet diet versus those fed an all-mash diet. Since the broilers raised on the crumbled-pellet diet also had increased body weights, it was unclear whether the increase in SDS mortality was due to the pelleting or to increased growth rate. Proudfoot et al. (1982) reported no significant differences in growth rates of broilers fed ground crumbled-pellet diets as compared with those fed all-mash diets yet the incidence of SDS was higher in broilers fed the former. They therefore concluded that the higher incidence of SDS
associated with crumbled-pellet diets was due to some factor(s) in the pelleting process rather than the increased growth rate resulting from the higher density of pelleted feeds.

Proudfoot et al. (1984) demonstrated that the incidence of SDS was significantly reduced when the dietary protein supplements of soybean meal, canola meal and fishmeal bypassed the pelleting process. There was no reduction in SDS mortality when either the micronutrient or fat components bypassed the pelleting process. They hypothesized that a toxic factor(s) was produced when protein supplements are subjected to pelleting, which may be involved in causing SDS.

2.2.9 Acid-base balance: Summers et al. (1987) have shown that lactic acid and/or a disruption of acid-base balance may be involved in the etiology of SDS. Lactic acid is produced in the chicken as a waste product of anaerobic oxidation and is produced in large quantities when white muscles are active. Lactic acid is also a fermentation product which is produced in the crop. The level of lactic acid in the crop depends upon the amount and type of feed present and the length of time the feed remains in the crop. Chickens fed mash diets have been shown to have a crop lactic acid concentration three times higher than that of birds maintained on a pelleted diet. The lactic acid produced in the crop is absorbed into the blood system (Bolton, 1965).

Summers et al. (1987) pipetted 5 ml of a 20% lactic acid solution into the crop of 2 week old male broiler chickens maintained on a regular broiler starter diet. Within a few minutes the broilers flipped in a manner similar to those
diagnosed as being affected by SDS. Broilers fed glucose supplemented diets were more prone to flip on lactic acid dosing than those fed a starch or fat supplemented diet.

Julian and Bowes (1987), however, observed that blood lactate levels were high in some broilers not affected by SDS, but were not elevated in broilers that had died from SDS.

2.2.10 Dietary additives: Proudfoot and Hulan (1983) evaluated the effects of aspirin (ASA) as a prophylactic drug to reduce the incidence of SDS in broilers. No beneficial effect on the incidence of SDS was observed and total mortality was increased. The addition of ASA at .16% of the diet resulted in decreased body weights.
3.0 LITERATURE REVIEW – TAURINE:

3.1 Introduction: Taurine was first isolated from ox bile in 1827 (Tiedemann and Gmelin, 1827) and for many years was believed to be solely an excretory product of sulfur amino acid metabolism. It has since been shown to be a major component of the free amino acid pool in animal body tissues (Jacobsen and Smith, 1968).

Structurally taurine ($H_2N-CH_2-CH_2-SO_3H$) is a $\beta$-sulphonic acid while the more common amino acids are $\alpha$-carboxylic acids. The sulphonic acid ($pK = 1.5$) and ammonium ($pK = 8.7$) functions of taurine are more acidic than the carboxylic acid ($pK = 2$) and ammonium ($pK = 9$) functions of $\alpha$-amino acids. As a result, taurine forms less stable complexes with the various transitional metals (Wright et al., 1986). The ability of amino acids to form metal complexes is an important feature of their biological activity.

Muscle contains the bulk of body taurine, especially red muscle which, in the chicken, has concentrations twenty times those found in white muscle ($21.3 \pm 3.7$ and $1.1 \pm .3$ umole/g wet weight, respectively) (Airaksenen and Partanen, 1985). The concentrations found in animal tissues vary between organs and between species. For example, levels of taurine in the heart vary from 40 umoles per gram wet weight for the adult rat to 4 umoles per gram wet weight for the cow (Huxtable, 1978). In general, the heart has the highest taurine concentrations and the liver shows the greatest amount of variation (Awapara et al., 1950).

Although taurine is widespread in animals, it has a very
limited distribution in plants (Jacobsen and Smith, 1968; Kataoka and Ohnishi, 1986). Taurine, or taurine derivatives, have been identified in some marine species of algae and some fungi. In the higher plants, taurine was identified in the pollen of some dicotyledons while no taurine has been detected in others (Jacobsen and Smith, 1968). When taurine is present in plants it is at concentrations about 1% of those found in animal tissues (Kataoka and Ohnishi, 1986).

3.2 Functions of Taurine: The first clearly recognized function of taurine was bile acid conjugation. Glycine and taurine bile salts aid in the emulsification of dietary lipids. This increases the surface area available for attack by the digestive enzymes and facilitates absorption of the free fatty acids. Taurine bile salts have two advantages over glycine bile salts in that they are more resistant to microbial degradation and they aid in the absorption of long chain saturated fatty acids (anonymous, 1988).

There is considerable species differences in regard to which of the two amino acids (glycine or taurine) is used for bile acid conjugation (Sturman and Hayes, 1980). In general, herbivores use predominantly glycine while carnivores used mainly taurine. The bile of the domestic fowl, an omnivore, contains both taurine and glycine bile salts (Duke, 1988).

Taurine also appears to play a role in regulating intracellular osmotic pressure. This is particularly evident in marine invertebrates. Surveys on the amino acid content of marine, brackish- and fresh-water crustaceans have shown that taurine concentrations are higher in the marine species (Allen
and Garrett, 1971). Taurine also appears to play a role in regulating intracellular osmotic pressure in the higher animals (van Gelder and Barbeau, 1985).

Interest in taurine increased when it was shown to be an essential nutrient for cats. Cats raised on a taurine-free diet developed retinal degeneration leading to blindness (Berson et al., 1976). It now appears that taurine is essential for maintaining the structural and functional integrity of photoreceptors, outer segments and tapetum lucidum of the eye (Huxtable and Sebring, 1976).

Taurine has recently been shown to be involved in a wide variety of phenomena. For example, taurine has been shown to act as an anti-convulsant and has been beneficial in the treatment of epilepsy (Koivisto et al., 1986). It is believed to act as a neuromodulator by stabilizing excitable membranes and by suppressing the release of neurotransmitters at synapses (Kuriyama, 1980). Taurine also appears to potentiate the effects of insulin since, in the presence of insulin, it stimulated glycolysis and glycogenesis (Lampson et al., 1983).

3.3 Sources of taurine: Tissue taurine is derived from either exogenous or endogenous sources. The relative importance of the two sources varies between species and appears to be related to diet. In general, carnivores, which have a large daily supply of dietary taurine, have little or no capacity for taurine biosynthesis while herbivores, which receive little or no taurine in the diet, are presumably able to synthesize all their own taurine (Huxtable and Lippincott, 1982).

Species differ in their ability to synthesize taurine from
various precursors. Five main pathways have been identified (see figure 3.1). They are summarized as follows:

Pathway I: methionine-cysteine-cysteine sulfinic acid-hypotaurine-taurine
Pathway II: methionine-cysteine-cysteine sulfinic acid-cysteic acid-taurine
Pathway III: cysteamine/cystamine-intermediates-hypotaurine-taurine
Pathway IV: sulfate-sulfite-intermediates-cysteic acid-taurine
Pathway V: cystine-cystine disulfioxide-cystamine disulfioxide-hypotaurine-taurine

In the domestic fowl taurine is synthesized by at least three different pathways (Jacobsen and Smith, 1968). The chick embryo liver is capable of producing taurine from methionine and cystine with cysteine sulfinic acid (CSA) and cysteic acid (CA) as intermediates. No hypotaurine formation was observed, indicating that CA was decarboxylated while CSA was not. It was thus concluded that in the embryonic liver taurine is synthesized via pathway II and that pathway I does not operate. The posthatch chick liver decarboxylase also has a higher affinity for CA than CSA indicating that taurine synthesis proceeds via pathway II. There is, however, slight but definite CSA-decarboxylase activity indicating that pathway I also operates, but to a limited extent. It was not clear whether the chick liver contained CSA specific decarboxylases not present in the embryonic liver, in addition to a CA-specific decarboxylase, or whether it was one enzyme which preferentially acts on CA but which can decarboxylate CSA to a limited extent.

In the chicken brain a different situation has been observed in that there appears to be a higher activity of CSA decarboxylation (to hypotaurine) than CA decarboxylation (Jacobsen et al., 1964). Thus, in the chicken brain, biosynthesis of taurine
Figure 3.1 Metabolic pathways related to taurine biosynthesis

\[
\begin{align*}
\text{METHIONINE} & \quad \rightarrow \\
\text{HOMOCYSTEINE} & \quad \leftrightarrow \quad \text{HOMOCYSTINE} \\
\text{serine} & \quad \rightarrow \\
\text{CYSTATHIONINE} & \quad \rightarrow \quad \text{HOMOCYSTINE} \\
\text{homoserine} & \quad \rightarrow \\
\text{CYSTEINE} & \quad \rightarrow \quad \text{CYSTEAMINE} \quad \equiv \quad \text{CYSTAMINE} \\
\text{CYSTINE} & \quad \rightarrow \\
\text{CYSTEINE} & \quad \rightarrow \quad \text{DISULFOXIDE} \\
\text{CO}_2 & \quad \downarrow \\
\text{CYSTAMINE} & \quad \rightarrow \quad \text{DISULFOXIDE} \\
\text{CYSTEINE} & \quad \rightarrow \quad \text{SULFINIC ACID} \quad \rightarrow \quad \text{HYPOTAURINE} \\
\text{CO}_2 & \quad \downarrow \\
\text{CYSTEIC ACID} & \quad \rightarrow \quad \text{Taurine} \\
\text{CO}_2 & \quad \downarrow \\
\text{serine} & \quad \rightarrow \\
\text{SULFITE} & \quad \leftrightarrow \quad \text{SULFATE}
\end{align*}
\]
appears to follow pathway I.

Chicken tissues have also been shown to synthesize taurine from inorganic sulfate following pathway IV (Machlin et al., 1955; Martin, 1972). The sulfate is reduced to sulfite which is then used together with L-serine in the production of L-cysteic acid. Pathway IV may be important in taurine production in the heart.

In the stressed heart transport appears to be more important than biosynthesis in the regulation of cardiac taurine levels. The rate of biosynthesis of taurine in the rat is constant. With increasing dietary taurine content the half-life of endogenous taurine decreases (Huxtable and Lippincott, 1982). The increase in taurine content associated with congestive heart failure appears to be due to an increase in taurine influx with no alteration in taurine biosynthesis (Huxtable, 1980).

3.4 Taurine and the heart: Taurine appears to play a role in regulating heart function but it is unclear what that function is. It is present in large concentrations in heart muscle and may represent more than 50% of the free amino acid pool (Kocsis et al., 1976). Myocardial metabolism of rats was found to be significantly altered by a depletion of cardiac taurine (Mozaffari et al., 1986) and taurine deficient cats have been shown to suffer from myocardial failure (Pion et al., 1987). Taurine has also been shown to have pharmacological value in the treatment of congestive heart failure (Azuma et al., 1985).

Read and Wetly (1963) showed that taurine administration (.5 mmol/kg body weight) prevented the development of
epinephrine and digoxin induced arrhythmias in dogs. Higher
doses (2 to 10 mmole/kg body weight) reversed existing
arrhythmias.

Kramer et al. (1978) noted that taurine protected the rat
heart against calcium paradox. Calcium paradox refers to the
phenomenon that occurs when a heart is perfused with calcium-
free medium and then reexposed to physiological calcium
concentrations. The zero calcium perfusion results in changes
to membrane ultrastructure and permeability so that reexposure
to calcium leads to calcium overload. Extensive cellular and
functional damage result. The presence of taurine in the
calcium-free medium prevents the loss of permeability and, thus
the calcium overload and cellular damage that occurs on
reexposure. Taurine appears to be acting as a membrane
stabilizer (Huxtable and Bressler, 1973) and may be modulating
ion fluxes (Hayes, 1976; Huxtable and Sebring, 1986).

3.5 Taurine and cardiomyopathy in poultry: Furazolidone
\((C_8H_7N_3O_2)\) is an antibacterial and antiprotozoan agent added to
poultry feeds (recommended dose is 110 mg/kg) to treat a number
of diseases. In turkey poult's the incidence of cardiomyopathy
has been shown to increase with furazolidone dose (Czarnecki et
al., 1974). The furazolidone induced cardiomyopathies resembled
the spontaneous round-heart syndrome and involved damage to the
myocardial cells (Czarnecki, 1980) and alteration of glycogen
metabolism (Czarnecki and Evanson, 1980). Furazolidone treated
poults had significantly \((p<.001)\) reduced cardiac taurine
content but brain and muscle levels were unaffected (Schaffer et
al., 1982). Oral administration of taurine to turkeys not
treated with furazolidone significantly (p<.001) increased taurine levels of the brain, muscle and heart. Oral administration of taurine to furazolidone treated turkeys restored myocardial taurine to normal levels but did not alter the incidence of furazolidone induced cardiomyopathies (Schaffer et al., 1982).

3.6 Reducing tissue taurine content: Taurine is taken up be a saturable active transport system. Hypotaurine, $\beta$-alanine, and guandinoethyl sulfonate (GES) are structural analogs of taurine and have been shown, in vitro, to be competitive inhibitors of taurine transport (Azari et al., 1979). Hypotaurine exists naturally in cells as an intermediate in taurine synthesis and would not be expected to be an effective taurine transport inhibitor in vivo. Transport of $\beta$-alanine into the heart has both a saturable and a nonsaturable component while GES is transported by a saturable process only (Huxtable et al., 1981).

GES is a naturally occurring substance found in the muscle tissue and gastrointestinal tract of some invertebrates. GES was not detected in the brain, blood or heart of rats, but was detected at low levels in the liver, kidney and muscles (Guiditto and Costagli, 1970).

A marked decrease in in vivo tissue taurine content in rats can be achieved by supplementing the drinking water with 1% GES (Huxtable et al., 1979). Cardiac taurine levels were reduced to 20-30% of normal while the levels of the other amino acids remained unaffected. The irreducible portion of the cardiac taurine content was due to biosynthesis within the heart. This
irreducible portion will vary between species depending upon the capacity for taurine synthesis.

The ability of GES to deplete tissue taurine content shows considerable species variation (Huxtable and Lippincott, 1981). Rats and mice do not metabolize GES so that administration of 1% GES in the drinking water of rats or mice fed taurine free diets causes large decreases in tissue taurine concentrations within a few days. GES administration has no effect on taurine levels in the guinea pig since this animal is herbivorous and would, presumably, not be dependent on the diet as a source of taurine. Cats fed taurine free diets and given 1% GES in the water do not have a decrease in tissue taurine levels since they are able to metabolize GES to taurine.

3.7 Taurine supplementation in broiler diets: Miller et al. (1987) supplemented the diet of male broiler chickens with taurine (0 to .6%) and reported that taurine supplementation had no effect on growth rate or feed efficiency. Any effects on mortality were not reported. Campbell and Classen (unpublished data) also supplemented the diets of male broiler chickens with taurine (0 to .2%). They found that taurine supplementation did not affect growth rate but improved feed efficiency. There was also some evidence that total mortality and mortality due to SDS were reduced by taurine supplementation.
4.0 EFFECT OF DIETARY LACTATE AND GLUCOSE CONTENT ON THE INCIDENCE OF SDS IN MALE BROILER CHICKENS

4.1 ABSTRACT:

A factorial experiment using 4 levels of dietary lactate (0, 2.5, 5.0, 7.5% calcium lactate) and 4 levels of dietary glucose (0, 15, 30 and 45% cerelose) was conducted to determine the effect of these compounds on the incidence of Sudden Death Syndrome (SDS) in male broiler chickens. A total of 1280 male broilers were reared in battery brooder cages to four weeks of age. Mean body weights and feed consumption were measured at two and four weeks of age. Mortality was recorded daily and any SDS affected birds identified by necropsy.

Overall mortality was 6.64% of which 32.9% (2.19% of broilers housed) was attributed to SDS. There were no significant differences between treatments in either total mortality or mortality due to SDS.

Feed consumption was influenced by the dietary level of both lactate and glucose. Inclusion of greater than 2.5% lactate in the diet reduced feed consumption while the inclusion of dietary glucose at 15% or higher increased feed consumption. There was a significant lactate x glucose interaction affecting 4 week mean body weights (p<.05). In general, as dietary lactate concentrations increased mean body weight was depressed. Conversely, mean body weights were higher as glucose content was increased. The depressing effect of dietary lactate was greatest when glucose was absent from the diet. Feed to gain ratios were significantly (p<.05) reduced by dietary lactate levels of 5.0 and 7.5% and increased by dietary glucose levels of 30 and 45%.
4.2 INTRODUCTION:

Sudden Death Syndrome (SDS) is a condition in which apparently healthy, fast growing broiler chicks die suddenly from no apparent cause. There is usually a short wing-beating convulsion prior to death so that the majority of affected broilers are found dead lying on their backs. As a result, the condition is often referred to as "Flip-Over disease". It has also been called "Acute Death Syndrome", "heart attack", "lung edema" and "died in good condition" (Merck, 1986).

SDS is a major cause of mortality in Canadian broiler flocks with individual incidences ranging from .71 to 4.07% of broilers housed (Riddell and Spring, 1985). Broilers of all ages are affected starting as early as two days of age and continuing through to market age. Peak mortality usually occurs between three and four weeks of age (Bridgen and Riddell, 1975; Ononiwu et al., 1979b; Gardiner et al., 1988b). The condition appears to be influenced by the sex of the chickens since more than 70% of SDS affected broilers are male (Hemsley, 1965; Brigden and Riddell, 1975; Steele and Edgar, 1982; Riddell and Springer, 1985).

The etiology of SDS is unknown, but it is generally believed to be a metabolic disorder (Julian and Bowes, 1987). Death is believed to be due to left ventricular fibrillation (Julian and Bowes, 1987), but it is unclear as to what causes this fibrillation to occur.

Julian and Leeson (1985) reported that the incidence of SDS was doubled in broilers fed glucose monohydrate based diets as compared to broilers fed corn or tallow based diets. They
hypothesized that SDS is related to a problem in carbohydrate metabolism.

Summers et al. (1987) noted that an SDS type death could be induced by injecting a 20% lactic acid solution into the wing vein of male broiler chicks. Pipetting 5 ml of the same 20% lactic acid solution into the crop of male broilers had variable results. Broilers receiving diets high in glucose "flipped" within 30 minutes of dosing with lactic acid while those broilers receiving a diet high in corn starch took over an hour and a half to "flip". It was therefore hypothesized that lactic acid or acid-base balance is involved in the etiology of SDS. Julian and Bowes (1987), however, found that blood lactate levels were high in some broilers unaffected by SDS, but were not elevated in broilers that died from SDS.

The purpose of this experiment was two-fold. First, to test the hypothesis that lactic acid is involved in the etiology of SDS and, second, to determine whether the level of dietary glucose would modify the effects of the lactic acid.

4.3 MATERIALS AND METHODS:

4.3.1 Design and treatments: Day-old commercial male broiler chicks (Hubbard) vaccinated against Marek's disease were placed on dietary treatment and reared in Petersime battery brooder cages to 4 weeks of age. The chicks were wing-banded and randomly allotted to 128 pens. A stocking rate of 10 broilers per pen was used, giving a total of 1280 chicks. The pens were 68 x 99 cm in size, giving a space allotment of .07 m²/broiler or 14.9 broilers/m².
A 4x4 factorial design was used and there were 4 blocks with 2 replications per block.

The broilers were fed isonitrogenous diets containing corn starch, corn oil, soybean meal and herring meal. Dietary calcium lactate levels varied from 0 to 7.5% and cerelose (glucose) levels from 0 to 45%, both at the expense of corn starch. Limestone content was adjusted to maintain dietary calcium concentrations of 1.35%. The diet compositions are shown in table 4.1. All diets were fed in mash form and each diet was fed to 8 pens (2 pens randomly selected in each of the 4 blocks).

4.3.2 Management: The broilers received 23 hours of light per day. Water and feed were available ad libitum. Mean body weights and feed consumption were measured on a pen basis at 2 and 4 weeks of age.

Initial brooder temperature was set at 35°C and reduced by 2.5°C weekly. At 3 weeks of age the brooder heaters were turned off.

Pens were checked twice daily for mortality and all dead broilers were necropsied. Death was attributed to SDS if there was no evidence of other disease and the broilers were in good body condition with a full digestive tract, an empty or small gall bladder and contracted ventricles.

4.3.3 Statistical analysis: Performance and mortality data were statistically analyzed using three-way analysis of variance procedures of the Statistical Analysis System (SAS Institute Inc., 1982). Any significant differences were further analyzed using the Ryan-Einot-Gabriel-Welsch multiple range test (SAS...
### Table 4.1. Composition and calculated analysis of diets used in Experiment 1

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<td>Limestone</td>
<td>2.7</td>
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<td>1.8</td>
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<td>0.9</td>
<td>0.9</td>
<td>0.9</td>
<td>0.9</td>
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</tr>
<tr>
<td>Sodium phosphate (monobasic)#</td>
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<td>1.5</td>
<td>1.5</td>
<td>1.5</td>
<td>1.5</td>
<td>1.5</td>
<td>1.5</td>
<td>1.5</td>
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<td>1.5</td>
<td>1.5</td>
<td>1.5</td>
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</tr>
<tr>
<td>Premix</td>
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<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
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<td>1.0</td>
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<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>D,L-Methionine</td>
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<td>0.2</td>
<td>0.2</td>
<td>0.2</td>
<td>0.2</td>
<td>0.2</td>
<td>0.2</td>
<td>0.2</td>
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<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>

Calculated analysis:

| Metabolizable energy (ME), kcal/kg | 3226 | 3223 | 3220 | 3217 | 3168 | 3165 | 3162 | 3159 | 3151 | 3107 | 3104 | 3101 | 3051 | 3048 | 3045 | 3042 |
| Crude Protein, %                  | 22.5 | 22.5 | 22.5 | 22.5 | 22.5 | 22.5 | 22.5 | 22.5 | 22.5 | 22.5 | 22.5 | 22.5 | 22.5 | 22.5 | 22.5 | 22.5 |
| Linoleic Acid, %                  | 1.54 | 1.54 | 1.54 | 1.54 | 1.54 | 1.54 | 1.54 | 1.54 | 1.54 | 1.54 | 1.54 | 1.54 | 1.54 | 1.54 | 1.54 | 1.54 |
| Meth. & Cys. (%)                  | 0.94 | 0.94 | 0.94 | 0.94 | 0.94 | 0.94 | 0.94 | 0.94 | 0.94 | 0.94 | 0.94 | 0.94 | 0.94 | 0.94 | 0.94 | 0.94 |
| Lysine, %                         | 1.58 | 1.58 | 1.58 | 1.58 | 1.58 | 1.58 | 1.58 | 1.58 | 1.58 | 1.58 | 1.58 | 1.58 | 1.58 | 1.58 | 1.58 | 1.58 |
| Ca, %                            | 1.35 | 1.35 | 1.35 | 1.35 | 1.36 | 1.36 | 1.36 | 1.36 | 1.36 | 1.36 | 1.36 | 1.36 | 1.36 | 1.36 | 1.36 | 1.36 |
| P, available, %                   | 0.54 | 0.64 | 0.64 | 0.64 | 0.64 | 0.64 | 0.64 | 0.64 | 0.64 | 0.64 | 0.64 | 0.64 | 0.64 | 0.64 | 0.64 | 0.64 |

Premix provided the following per kg diet: 9,000 IU vitamin A; 1,500 IU vitamin D3; 10 IU vitamin E; 0.5 mg vitamin K; 0.007 mg vitamin B12; 0.4 mg thiamin; 6 mg riboflavin; 1 mg folic acid; 0.15 mg biotin; 12 mg pantothenic acid; 35 mg niacin; 4 mg pyridoxine; 1000 mg choline; 1,184 mg methionine; 0.125 mg ethoxyquin; 2 g salt; 60 mg manganese; 5 mg copper; 50 mg zinc; 0.1 mg selenium; 0.35 mg iodine.

* 47% crude protein

® 26% P and 20% Na
Institute Inc., 1982). An arcsin transformation was used before analyzing the mortality data, but for interpretation purposes the results are presented as percentages.

4.4 RESULTS:

Similar results were obtained at 2 and 4 weeks of age so only the 4 week data are presented. Total mortality (table 4.2) was 6.64% of which 32.9% was attributed to SDS. Twenty-seven percent of the total mortality occurred during the first week and was attributed principally to omphalitis and starve-outs. There were no statistically significant differences between treatments for the incidence of SDS expressed as a percent of broilers housed (table 4.3) or as a percent of total mortality (table 4.4).

Feed consumption, mean body weight and feed conversion at 4 weeks of age are shown in tables 4.5 to 4.7. Feed consumption was significantly (p<.05) affected by both dietary lactate and glucose concentrations. As lactate concentrations increased above 2.5%, feed consumption declined. Conversely, feed consumption increased as glucose content increased. There was a significant (p<.05) lactate x glucose interaction affecting final body weight. This is depicted in figures 4.1 and 4.2. The effect of dietary lactate supplementation was dependent on the level of dietary glucose, but, in general, inclusion of dietary lactate decreased, while dietary glucose increased, mean body weight. The depressing effect of dietary lactate was most dramatic when there was no glucose in the diet. Feed conversion (g feed/g body weight gain) was significantly (p<.05) reduced by
Table 4.2 Effect of dietary lactate and glucose on total mortality (Experiment 1): Results at 4 weeks of age

<table>
<thead>
<tr>
<th>LACTATE (% of diet)</th>
<th>GLUCOSE (% of diet)</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>15</td>
</tr>
<tr>
<td>0.0</td>
<td>9</td>
<td>11.25</td>
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<tr>
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<td>3</td>
<td>3.75</td>
</tr>
<tr>
<td>TOTAL</td>
<td>21</td>
<td>6.56</td>
</tr>
</tbody>
</table>

1 Percent of broilers housed (80 per dietary treatment)
Note: No significant differences between dietary treatments
Table 4.3  Effect of dietary lactate and glucose on SDS mortality as a percent of broilers housed (Experiment 1): Results at 4 weeks of age

<table>
<thead>
<tr>
<th>LACTATE (% of diet)</th>
<th>0</th>
<th>15</th>
<th>30</th>
<th>45</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>GLUCOSE (% of diet)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. %</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>6</td>
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<tr>
<td></td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>7</td>
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<td>1</td>
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<td>2</td>
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<td>11</td>
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<td>0</td>
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<td>2</td>
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<td>4</td>
</tr>
<tr>
<td>TOTAL</td>
<td>6</td>
<td>6</td>
<td>5</td>
<td>11</td>
<td>28</td>
</tr>
</tbody>
</table>

1 Percent of broilers housed (80 per dietary treatment)

Note: No significant differences between dietary treatments
Table 4.4 Effect of dietary lactate and glucose on SDS mortality as percent of total mortality (Experiment 1): Results at 4 weeks of age

<table>
<thead>
<tr>
<th>LACTATE (% of diet)</th>
<th>0</th>
<th>15</th>
<th>30</th>
<th>45</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.0</td>
<td>33.3</td>
<td>11.1</td>
<td>0.0</td>
<td>40.0</td>
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<td>0.0</td>
<td>0.0</td>
<td>40.0</td>
<td>40.0</td>
<td>26.7</td>
</tr>
</tbody>
</table>

TOTAL 28.6 28.6 26.3 45.8 32.9

Note: No significant differences between dietary treatments
Table 4.5 Effect of dietary lactate and glucose on mean total feed consumption (g/broiler) (Experiment 1): Results at 4 weeks of age

<table>
<thead>
<tr>
<th>LACTATE (% of diet)</th>
<th>0</th>
<th>15</th>
<th>30</th>
<th>45</th>
<th>AVE:</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.0</td>
<td>1173 ± 25</td>
<td>1294 ± 27</td>
<td>1357 ± 32</td>
<td>1426 ± 32</td>
<td>1312 ± 22&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>2.5</td>
<td>1065 ± 32</td>
<td>1290 ± 25</td>
<td>1334 ± 39</td>
<td>1443 ± 14</td>
<td>1283 ± 28&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>5.0</td>
<td>944 ± 16</td>
<td>1187 ± 41</td>
<td>1305 ± 31</td>
<td>1347 ± 45</td>
<td>1196 ± 33&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>7.5</td>
<td>883 ± 36</td>
<td>1031 ± 38</td>
<td>1236 ± 36</td>
<td>1257 ± 18</td>
<td>1102 ± 32&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

AVE: 1016 ± 24<sup>a</sup> 1201 ± 25<sup>b</sup> 1308 ± 18<sup>c</sup> 1368 ± 19<sup>d</sup> 1223 ± 16

Data shown as mean ± SEM

a, b, c, d Means within a row or column with different superscripts are significantly (p<.05) different
Table 4.6 Effect of dietary lactate and glucose on mean body weight (g/broiler) at 4 weeks of age (Experiment 1)

<table>
<thead>
<tr>
<th>LACTATE (% of diet)</th>
<th>GLUCOSE (% of diet)</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.0</td>
<td>878 ± 20&lt;sup&gt;bc&lt;/sup&gt; 922 ± 18&lt;sup&gt;ab&lt;/sup&gt; 948 ± 26&lt;sup&gt;ab&lt;/sup&gt; 981 ± 24&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>932 ± 13</td>
</tr>
<tr>
<td>2.5</td>
<td>793 ± 27&lt;sup&gt;cd&lt;/sup&gt; 941 ± 19&lt;sup&gt;ab&lt;/sup&gt; 954 ± 29&lt;sup&gt;ab&lt;/sup&gt; 998 ± 19&lt;sup&gt;a&lt;/sup&gt;</td>
<td>922 ± 18</td>
</tr>
<tr>
<td>5.0</td>
<td>734 ± 12&lt;sup&gt;de&lt;/sup&gt; 876 ± 27&lt;sup&gt;bc&lt;/sup&gt; 929 ± 24&lt;sup&gt;ab&lt;/sup&gt; 962 ± 11&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>876 ± 18</td>
</tr>
<tr>
<td>7.5</td>
<td>665 ± 22&lt;sup&gt;e&lt;/sup&gt; 786 ± 23&lt;sup&gt;cd&lt;/sup&gt; 886 ± 20&lt;sup&gt;bc&lt;/sup&gt; 903 ± 18&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>810 ± 20</td>
</tr>
<tr>
<td>TOTAL</td>
<td>768 ± 17 882 ± 15 930 ± 13 961 ± 11</td>
<td>885 ± 10</td>
</tr>
</tbody>
</table>

Data shown as mean ± SEM
Significant glucose x lactate interaction (p<.05)
a,b,c,d,e Means with different superscripts are significantly (p<.05) different
Table 4.7  Effect of dietary lactate and glucose on mean feed conversion (g feed/g body weight gain) from 0 to 4 weeks of age (Experiment 1)

<table>
<thead>
<tr>
<th>LACTATE (% of diet)</th>
<th>GLUCOSE (% of diet)</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.0</td>
<td>1.425 + .016</td>
<td>1.480 + .010&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>2.5</td>
<td>1.420 + .013</td>
<td>1.459 + .011&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>5.0</td>
<td>1.368 + .012</td>
<td>1.432 + .014&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>7.5</td>
<td>1.420 + .011</td>
<td>1.434 + .010&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Data shown as mean + SEM

<sup>a,b</sup> Means within a row or column with different superscripts are significantly different (p<.05)
Figure 4.1 Effects of dietary lactate, at different levels of dietary glucose, on mean body weight at 4 weeks of age (Experiment 1)
Figure 4.1 Effects of dietary lactate, at different levels of dietary glucose, on mean body weight at 4 weeks of age (Experiment 1)
Figure 4.2 Effect of dietary glucose, at different levels of dietary lactate, on mean body weight at 4 weeks of age (Experiment 1)
Figure 4.2 Effect of dietary glucose, at different levels of dietary lactate, on mean body weight at 4 weeks of age (Experiment 1)
inclusion of 5 or 7.5% lactate. Inclusion of 30 or 45% glucose significantly (p<.05) increased feed conversion.

4.5 DISCUSSION:

Summers et al. (1987) induced an SDS-like death by injecting a 20% lactic acid solution into the wing vein of male broiler chickens. Pipetting 5 ml of the same lactic acid solution into the crop produced variable results depending on the previous diet of the broiler. Those reared on glucose based diets "flipped" within 30 minutes of dosing while those on corn starch based diets took over an hour and a half to die. It was therefore hypothesized that lactic acid was involved in the etiology of SDS. The results of this experiment, however, do not support this hypothesis. Supplementation with calcium lactate to 7.5% of the diet of male broiler chickens had no effect on total mortality or mortality due to SDS. The level of dietary glucose had no effect on the incidence of SDS and did not modify the effects of calcium lactate supplementation. The level of lactic acid used by Summers et al. (1987) to produce the SDS-like death is probably in excess of what would normally occur in the crop of broilers. It is therefore unlikely that lactic acid is involved in the etiology of SDS.

Growth rate was significantly depressed by lactic acid supplementation with no effect on SDS mortality. Thus the results of this experiment do not support the hypothesis of Bowes et al. (1988) that there is a relationship between growth rate and the incidence of SDS.
5.0 EFFECT OF DIETARY PROTEIN SOURCE AND CEREAL TYPE ON THE INCIDENCE OF SDS IN MALE BROILER CHICKENS

5.1 ABSTRACT:

Three experiments were conducted to compare the incidence of SDS, growth rate and feed conversion for male broiler chickens fed isocaloric and isonitrogenous diets with either corn or wheat as the grain type and meat meal or soybean meal as the main protein source. In the first two experiments the broilers were raised in floor pens to 6 weeks of age while in the third experiment the broilers were raised in battery brooder cages to 4 weeks of age. All broilers were Peterson x Arbor Acre.

The effect of cereal type on mortality, mean body weight and feed conversion was inconsistent over the three studies. In both floor pen studies total mortality and the incidence of SDS were significantly higher for wheat-fed broilers while SDS as a percent of broilers housed was not affected by cereal type. In the brooder study neither total mortality nor SDS mortality was significantly affected by cereal type. Final mean body weights were significantly higher for the wheat-fed broilers in the first floor pen study (p<.01) and in the brooder study (p<.01). In the second floor pen study, however, cereal type did not have a significant effect on mean body weight. There was a significant (p<.05) cereal x protein interaction affecting feed conversion in the two floor pen studies, but in the brooder study feed conversion was not affected by either cereal type or protein source.

The effect of protein source on mortality, mean body weight and feed conversion was also inconsistent across the three
studies. In the floor pen studies the incidence of SDS, as a percent of broilers housed, was reduced by the inclusion of meat meal in the diet. In the first floor pen study total mortality was unaffected by protein source while SDS as a percent of total mortality was reduced. The reverse occurred in the second floor pen study in that total mortality was reduced by the inclusion of meat meal in the diet while SDS as a percent of total mortality was unaffected. In the brooder study total mortality and the incidence of SDS were not affected by protein source, but SDS as a percent of total mortality was reduced with the inclusion of meat meal in the diet. In the first floor pen study final mean body weight was significantly higher for the broilers receiving meat meal in the diet, but there was no effect of protein source on final mean body weight in the other two studies.

5.2 INTRODUCTION:

Sudden Death Syndrome (SDS) is a condition in which apparently healthy fast growing broiler chicks die suddenly from no apparent cause. There is usually a short, wing-beating convulsion prior to death so that the majority of affected broilers are found dead lying on their backs. As a result, the condition has often been referred to as "Flip-Over" disease. SDS has also been referred to as "Acute Death Syndrome", "heart attack", "lung edema", "died in good condition" and "fatal syncope" (Merck, 1986).

SDS is a major cause of mortality in Canadian broiler flocks with individual incidences ranging from 0.71 to 4.07% of broilers
housed (Riddell and Springer, 1985). Broilers of all ages are affected starting as early as two days of age and continuing through to market age with peak mortality usually occurring between three and four weeks of age (Brigden and Riddell, 1975; Ononiwu et al., 1979b; Gardiner et al., 1988b). The condition appears to be influenced by the sex of the chicken since more than 70% of SDS affected broilers are male (Hemsley, 1965; Brigden and Riddell, 1975; Steele and Edgar, 1982; Riddell and Springer, 1985).

The etiology of SDS is unknown, but it is generally believed to be a metabolic disorder (Julian and Bowes, 1987). Various dietary (Hulan et al., 1980; Hunt and Gardiner, 1982; Proudfoot et al., 1982; Steele et al., 1982; Mollison et al., 1984 and Wu and Nakaue, 1987) and management (Ononiwu et al., 1979b; Newberry et al., 1985 and Newberry et al., 1986) practices and a variety of chemicals (Proudfoot and Hulan, 1983 and Gardiner and Hunt, 1984) have been studied to try and alter the occurrence of SDS. The results have been inconsistent from study to study.

The involvement of dietary cereal type on the etiology of SDS is unclear. Hunt and Gardiner (1982) reported no differences in total or SDS mortality between flocks fed corn- or wheat-soy based diets. Mollison (1983), however, noted that a higher incidence of SDS was associated with wheat-soy based diets than with corn-soy based diets. In a later study, however, Mollison et al. (1984) showed that corn-fed broilers had significantly better weight gain and feed to gain ratios than wheat-fed groups but total and SDS mortality were not significantly affected by cereal type. Riddell and Springer (1985), in a survey of 51
broiler flocks in Alberta and Saskatchewan, reported a relationship between dietary cereal type and the incidence of SDS. The incidence of SDS was higher in flocks supplied by a feed company which used less corn, and more wheat, than the feed companies supplying the other broiler flocks surveyed.

Proudfoot et al. (1982) observed a higher incidence of SDS associated with pelleting of feed. When the dietary protein supplements by-passed the pelleting process the incidence of SDS was reduced (Proudfoot et al., 1984). They hypothesized that a toxic factor(s) was produced when protein supplements are subjected to pelleting, which may be involved in causing SDS. There was no comparison done on which type of protein supplement, animal or plant, was most affected by pelleting.

The purpose of the three experiments conducted was to compare the effect of cereal type (wheat versus corn) and protein source (soybean meal versus meat meal) on the incidence of SDS in male broiler chickens.

5.3 MATERIALS AND METHODS:

All three experiments used day-old commercial male broiler chicks (Peterson X Arbor Acre) vaccinated against Marek's disease.

5.3.1 Experiments 2 and 3 - Design and treatments: In Experiment 2, six thousand chicks were randomly distributed among 16 floor pens (4.95 x 8.59 m each) at a stocking density of 8.8 broilers/m² or .11 m²/broiler (375 broilers/pen). In Experiment 3, nine thousand and six hundred chicks were randomly distributed among the same 16 floor pens at a stocking density
of 14.1 broilers/m$^2$ or .07 m$^2$/broiler (600 broilers/pen). Wood shavings were used as litter.

Both experiments used a 2x2 factorial design with 4 blocks and 1 replication per block.

The broilers were fed either a corn or wheat based diet. Dietary protein was supplied as soybean meal with or without supplemental meat meal. A starter series of diets (22.5% CP) medicated with monensin sodium as the coccidiostat were fed from day-old to 21 days of age. A grower series of diets (21% CP) medicated with salinomycin as the coccidiostat were fed from 21 to 40 days of age. The compositions of the diets are shown in table 5.1. The starter diets were fed as crumbles and the grower diets as pellets (10 mm long x 5 mm in diameter). All diets were pelleted and crumbled using a steam-pressure-die process. Each diet was fed to four pens (1 pen per block).

5.3.2 Experiments 2 and 3 - Management: Feed and water were supplied ad libitum. At 21 and 40 days of age days pen feed consumption and body weights were recorded. A sample of approximately 75 broilers per pen was taken and the broilers weighed individually to calculate average pen body weight.

For the first week the chicks of each pen were kept within a 12 foot diameter confinement ring. Each pen was equipped with 1 gas brooder, 2 water founts, 2 feed trays and 6 tube feeders. At one week of age the confinement rings were removed. For the remainder of the experiment the broilers were provided with fifteen tube feeders and four 2.4 m automatic water troughs in each pen.

Initial brooder temperature was set at 35°C and was reduced
Table 5.1. Composition and calculated analysis of diets used in Experiments 2, 3 and 4

<table>
<thead>
<tr>
<th>Ingredients</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
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</thead>
<tbody>
<tr>
<td>Corn</td>
<td>58.8</td>
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<td>62.9</td>
<td>57.9</td>
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<td>Wheat</td>
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<td>57.3</td>
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<td>Soybean meal*</td>
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<td>1.3</td>
<td>1.9</td>
<td>1.3</td>
<td>1.9</td>
</tr>
<tr>
<td>Dical. phosphate#</td>
<td>0.0</td>
<td>1.7</td>
<td>0.0</td>
<td>1.4</td>
<td>0.0</td>
<td>1.7</td>
<td>0.0</td>
<td>1.7</td>
</tr>
<tr>
<td>D,L-Methionine</td>
<td>0.22</td>
<td>0.22</td>
<td>0.23</td>
<td>0.23</td>
<td>0.05</td>
<td>0.05</td>
<td>0.07</td>
<td>0.07</td>
</tr>
<tr>
<td>Premix</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Hardener</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Calculated analysis:

Metabolizable energy (ME),
kcal/kg 3100 3100 3100 3100 3100 3100 3100 3100
Crude Protein, % 22.5 22.5 22.5 22.5 21.0 21.0 21.0 21.0
Linoleic Acid, % 3.54 4.18 3.57 4.09 3.25 3.85 3.62 3.80
Methionine, % 0.59 0.59 0.56 0.56 0.40 0.40 0.38 0.38
Lysine, % 1.23 1.29 1.23 1.23 1.13 1.17 1.16 1.16
Ca, % 1.2 1.2 1.2 1.2 1.2 1.2 1.2 1.2
P, available, % 0.60 0.54 0.60 0.54 0.54 0.54 0.60 0.60

Premix provided the following per kg diet: 9,000 IU vitamin A; 1,500 IU vitamin D3; 10 IU vitamin E; 0.5 mg vitamin K; 0.007 mg vitamin B12; 0.4 mg thiamin; 6 mg riboflavin; 1 mg folic acid; 0.15 mg biotin; 12 mg pantothenic acid; 35 mg niacin; 4 mg pyridoxine; 1,000 mg choline; 0.125 mg ethoxyquin; 60 mg manganese; 5 mg copper; 50 mg zinc; 0.1 mg selenium; 0.35 mg iodine.

The starter premix also provided 99 mg/kg diet of monensin sodium as the coccidiostat while the finisher premix provided 60 mg/kg diet of salinomycin as the coccidiostat.

* 47% Crude protein
# 16% Ca; 20% P
by 2.5°C weekly. At 5 weeks of age the brooder heaters were turned off. Barn temperature was maintained at around 18.5°C (daily average of 18.9 ± 0.1 for blocks 1 and 2 and 18.1 ± 0.1 for block 3 and 4).

The broilers received 23 hours of light per day. Light intensity, measured 5 inches above the level of the shavings, was 7.5 lux from 1-4 days, 5 lux from 5-9 days, 3 lux from 10-14 days and 1 lux thereafter. Lights were brightened while checking for mortality and for feeding.

The broilers were checked twice daily for mortality and all dead broilers were necropsied. Death was attributed to SDS if there was no evidence of other disease and the broilers were in good body condition with a full digestive tract, a small or empty gall bladder and contracted ventricles.

Experiment 2 started in September of 1987 while Experiment 3 started in May of 1988.

5.3.3 Experiment 4 - Design and treatments: In Experiment 4, started in July of 1987, the same starter series of diets used in the floor pen studies were fed to chicks reared in Petersime battery brooder cages to four weeks of age. The design was a 2x2 factorial with 2 blocks and 8 replications per block. Six hundred and forty day-old chicks were randomly allotted to 64 pens to give a density of 10 broilers per pen. Each pen was 68 x 99 cm in size and therefore provided 0.07 m²/broiler. Each diet was fed to sixteen pens (8 pens per block).

5.3.4 Experiment 4 - Management: The broilers received 23 hours of light per day and were supplied feed and water ad libitum. Mean body weights and feed consumption were determined
Initial brooder temperature was set at 35°C and reduced by 2.5°C weekly. At 3 weeks of age the brooder heaters were turned off.

5.3.5 Statistical analysis: The performance and mortality data for all three experiments were analyzed using the analysis of variance procedures of the Statistical Analysis System (SAS Institute Inc., 1982). Any significant interactions were further analyzed using the Ryan-Einot-Gabriel-Welsch multiple range test. Mortality data were converted by an arcsin transformation before analysis but for interpretation purposes are presented as percentages.

5.4 RESULTS:

5.4.1 Experiment 2: The performance and mortality data of Experiment 2 at 21 and 40 days of age are shown in tables 5.2 and 5.3, respectively. At 21 days of age cereal type did not significantly affect total mortality, the incidence of SDS, SDS as a percent of total mortality, or feed conversion. Body weights, however, were significantly lower for the broilers fed the wheat-based diets. SDS mortality was significantly (p<.01) reduced by inclusion of meat meal in the diet, but there was no effect of protein source on total mortality, SDS as a percent of total mortality, body weight or feed conversion.

By 40 days of age grain source significantly (p<.05) affected both total mortality and SDS mortality expressed as a percent of broilers housed. A higher incidence of total and SDS mortality was associated with the wheat based diets.
Table 5.2 Effect of diet on final body weight, feed conversion and mortality (Experiment 2): Results at 21 days of age

<table>
<thead>
<tr>
<th>DIET</th>
<th>Final body weight (g/broiler)</th>
<th>Feed conversion (g feed/g gain)</th>
<th>Total Mortality</th>
<th>SDS Mortality</th>
<th>% of total mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>corn/meat meal</td>
<td>769 ± 2</td>
<td>1.278 ± .005</td>
<td>23</td>
<td>1.5</td>
<td>15</td>
</tr>
<tr>
<td>corn/SBM</td>
<td>744 ± 5</td>
<td>1.289 ± .007</td>
<td>30</td>
<td>2.0</td>
<td>25</td>
</tr>
<tr>
<td>wheat/meat meal</td>
<td>715 ± 9</td>
<td>1.304 ± .015</td>
<td>29</td>
<td>1.9</td>
<td>17</td>
</tr>
<tr>
<td>wheat/SBM</td>
<td>723 ± 4</td>
<td>1.276 ± .005</td>
<td>30</td>
<td>2.0</td>
<td>20</td>
</tr>
<tr>
<td>overall:</td>
<td>737 ± 6</td>
<td>1.287 ± .005</td>
<td>112</td>
<td>1.9</td>
<td>77</td>
</tr>
</tbody>
</table>

Main effects:

| corn             | 756 ± 5                       | 1.284 ± .005                   | 53              | 1.8           | 40                   | 1.3 | 75.5 |
| wheat            | 719 ± 5                       | 1.289 ± .009                   | 59              | 2.0           | 37                   | 1.2 | 62.7 |
| significance:    | *                             | NS                             | NS              | NS            | NS                   |     |     |
| meat meal        | 742 ± 11                      | 1.291 ± .009                   | 52              | 1.7           | 32                   | 1.1 | 61.5 |
| SBM              | 734 ± 5                       | 1.282 ± .005                   | 60              | 2.0           | 45                   | 1.5 | 75.0 |
| significance:    | NS                            | NS                             | NS              | NS            | **                   |     |     |

Data shown as mean ± SEM
* p<.05
** p<.01
1 Percent of broilers housed (1500/dietary treatment)
Table 5.3 Effect of diet on final body weight, feed conversion and mortality (Experiment 2): Results at 40 days of age

<table>
<thead>
<tr>
<th>DIET</th>
<th>Final body weight (g/broiler)</th>
<th>Feed conversion (g feed/g gain)</th>
<th>Total Mortality</th>
<th>SDS Mortality</th>
<th>SDS Mortality % of total mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>corn/meat meal</td>
<td>2130 ± 12</td>
<td>1.682 ± .013</td>
<td>46</td>
<td>28</td>
<td>1.8</td>
</tr>
<tr>
<td>corn/SBM</td>
<td>2112 ± 20</td>
<td>1.673 ± .009</td>
<td>56</td>
<td>43</td>
<td>2.9</td>
</tr>
<tr>
<td>wheat/meat meal</td>
<td>2061 ± 19</td>
<td>1.606 ± .013</td>
<td>80</td>
<td>46</td>
<td>3.1</td>
</tr>
<tr>
<td>wheat/SBM</td>
<td>1978 ± 14</td>
<td>1.648 ± .007</td>
<td>76</td>
<td>52</td>
<td>3.5</td>
</tr>
<tr>
<td>overall:</td>
<td>2070 ± 17</td>
<td>1.652 ± .009</td>
<td>258</td>
<td>169</td>
<td>2.8</td>
</tr>
</tbody>
</table>

Main effects:
- corn: 2121 ± 11, 1.677 ± .007, 102, 3.4, 71, 2.4, 69.6
- wheat: 2020 ± 19, 1.627 ± .011, 156, 5.2, 98, 3.3, 62.8

significance: ** *interaction *

meat meal: 2096 ± 17, 1.644 ± .017, 126, 4.2, 74, 2.5, 58.7
SBM: 2045 ± 28, 1.660 ± .007, 132, 4.4, 95, 3.2, 72.0

significance: * *interaction NS *

Data shown as mean ± SEM
* p<.05
** p<.01
1 Percent of broilers housed (1500/dietary treatment)
Figure 5.1 Effect of dietary cereal type on feed conversion (g feed/g body weight gain) at 40 days of age (Experiment 2)
Figure 5.2 Effect of dietary protein source on feed conversion (g feed/g body weight gain) at 40 days of age (Experiment 2)
There was no effect of cereal type on SDS as a percent of total mortality. The incidence of SDS was significantly (p<.05) affected by dietary protein source. SDS as a percent of total mortality and as a percent of broilers housed was increased when meat meal was excluded from the diet. There was no effect of dietary protein source on total mortality. Body weights were significantly affected by both cereal type (p<.01) and dietary protein source (p<.05). Broilers receiving meat meal in the diet weighed more than those receiving only SBM as the dietary protein source. Corn based diets supported a higher final body weight than wheat based diets. There was a significant (p<.05) cereal x protein interaction affecting feed conversion. This is shown graphically in figures 5.1 and 5.2. Broilers fed wheat based diets had lower feed conversion ratios than those fed corn based diets only when meat meal was included in the diet. The inclusion of meat meal in corn based diets did not significantly affect feed conversion ratios but inclusion of meat meal did significantly (p<.05) reduce feed conversion ratios of broilers fed wheat based diets.

5.4.2 Experiment 3: The performance and mortality data of Experiment 3 at 21 and 40 days of age are shown in tables 5.4 and 5.5, respectively. At 21 days of age the broilers fed the wheat based diets had significantly (p<.05) higher total mortality but the incidence of SDS was not significantly affected. There was no significant effect of cereal type on mean body weight or feed conversion. Protein source did not significantly affect total mortality or mortality due to SDS, but the inclusion of meat meal resulted in significantly (p<.05)
Table 5.4 Effect of diet on final body weight, feed conversion and mortality (Experiment 3): Results at 21 days of age

<table>
<thead>
<tr>
<th>DIET</th>
<th>Final body weight (g/broiler)</th>
<th>Feed conversion (g feed/g gain)</th>
<th>Total Mortality</th>
<th>SDS Mortality</th>
<th>SDS Mortality % of total mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>corn/meat meal</td>
<td>699 ± 7</td>
<td>1.325 ± 0.012</td>
<td>70 2.9</td>
<td>40 1.7</td>
<td>57.1</td>
</tr>
<tr>
<td>corn/SBM</td>
<td>744 ± 7</td>
<td>1.308 ± 0.007</td>
<td>74 3.1</td>
<td>45 1.9</td>
<td>60.8</td>
</tr>
<tr>
<td>wheat/meat meal</td>
<td>689 ± 6</td>
<td>1.344 ± 0.013</td>
<td>93 3.9</td>
<td>47 2.0</td>
<td>50.5</td>
</tr>
<tr>
<td>wheat/SBM</td>
<td>721 ± 13</td>
<td>1.313 ± 0.013</td>
<td>116 4.8</td>
<td>66 2.8</td>
<td>56.9</td>
</tr>
<tr>
<td>overall:</td>
<td>713 ± 7</td>
<td>1.323 ± 0.006</td>
<td>353 3.7</td>
<td>198 2.1</td>
<td>56.1</td>
</tr>
</tbody>
</table>

Main effects:
- corn
  - 721 ± 10
  - 1.317 ± 0.007
  - 144 3.0
  - 95 2.0
  - 66.0
- wheat
  - 705 ± 9
  - 1.329 ± 0.010
  - 209 4.4
  - 113 2.4
  - 54.1

Significance:
- NS
- NS
- *
- NS
- NS

meat meal
- 694 ± 5
- 1.335 ± 0.009
- 163 3.4
- 87 1.8
- 53.4

SBM
- 732 ± 8
- 1.311 ± 0.007
- 190 4.0
- 111 2.3
- 58.4

Significance:
- NS
- NS
- NS
- NS
- NS

Data shown as mean ± SEM
* p<.05
** p<.01
1 Percent of broilers housed (2400/dietary treatment)
Table 5.5 Effect of diet on final body weight, feed conversion and mortality (Experiment 3): Results at 40 days of age

<table>
<thead>
<tr>
<th>DIET</th>
<th>Final body weight (g/broiler)</th>
<th>Feed conversion (g feed/g gain)</th>
<th>Total Mortality</th>
<th>SDS Mortality</th>
<th>SDS Mortality % of total mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>corn/meat meal</td>
<td>1892 ± 25</td>
<td>1.810 ± .022</td>
<td>123 5.1</td>
<td>62 2.6</td>
<td>50.4</td>
</tr>
<tr>
<td>corn/SBM</td>
<td>1974 ± 16</td>
<td>1.790 ± .005</td>
<td>148 6.2</td>
<td>94 3.9</td>
<td>63.5</td>
</tr>
<tr>
<td>wheat/meat meal</td>
<td>1978 ± 5</td>
<td>1.767 ± .012</td>
<td>175 7.3</td>
<td>96 4.0</td>
<td>54.9</td>
</tr>
<tr>
<td>wheat/SBM</td>
<td>1976 ± 33</td>
<td>1.814 ± .018</td>
<td>227 9.5</td>
<td>131 5.5</td>
<td>57.7</td>
</tr>
<tr>
<td>overall:</td>
<td>1955 ± 14</td>
<td>1.795 ± .008</td>
<td>673 7.0</td>
<td>383 4.0</td>
<td>56.9</td>
</tr>
</tbody>
</table>

Main effects:
- corn
  - 1933 ± 21
  - 1.800 ± .011
  - 271 5.6
  - 156 3.3
  - 57.6
- wheat
  - 1977 ± 16
  - 1.790 ± .013
  - 402 8.4
  - 227 4.7
  - 56.5

Significance: NS *interaction ** NS

- meat meal
  - 1935 ± 20
  - 1.788 ± .014
  - 298 6.2
  - 158 3.3
  - 53.0
- SBM
  - 1975 ± 17
  - 1.802 ± .010
  - 375 7.8
  - 225 4.7
  - 60.0

Significance: NS *interaction * NS

Data shown as mean ± SEM
* p<.05
** p<.01
1 Percent of broilers housed (2400/dietary treatment)
Figure 5.3 Effect of dietary cereal type on feed conversion (g feed/g body weight gain) at 40 days of age (Experiment 3)

<table>
<thead>
<tr>
<th>Dietary cereal</th>
<th>Main dietary protein</th>
<th>Corn</th>
<th>Wheat</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>meat meal</td>
<td>1.81</td>
<td>1.80</td>
</tr>
<tr>
<td></td>
<td>SBM</td>
<td>1.78</td>
<td>1.76</td>
</tr>
</tbody>
</table>
Figure 5.4 Effect of dietary protein source on feed conversion (g feed/g body weight gain) at 40 days of age (Experiment 3)
Table 5.6 Effect of diet on final body weight, feed conversion and mortality (Experiment 4):
Results at 28 days of age

<table>
<thead>
<tr>
<th>DIET</th>
<th>Final body weight (g/broiler)</th>
<th>Feed conversion (g feed/g gain)</th>
<th>Total Mortality</th>
<th>SDS Mortality</th>
<th>SDS Mortality % of total mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>No. %</td>
<td>No. %</td>
<td></td>
</tr>
<tr>
<td>corn/meat meal</td>
<td>1123 ± 18</td>
<td>1.473 ± .011</td>
<td>10 6.3</td>
<td>4 2.5</td>
<td>40.0</td>
</tr>
<tr>
<td>corn/SBM</td>
<td>1121 ± 10</td>
<td>1.514 ± .012</td>
<td>7 4.4</td>
<td>6 3.8</td>
<td>85.7</td>
</tr>
<tr>
<td>wheat/meat meal</td>
<td>1071 ± 15</td>
<td>1.508 ± .014</td>
<td>9 5.6</td>
<td>2 1.2</td>
<td>22.2</td>
</tr>
<tr>
<td>wheat/SBM</td>
<td>1062 ± 14</td>
<td>1.513 ± .019</td>
<td>6 3.8</td>
<td>3 1.9</td>
<td>50.0</td>
</tr>
<tr>
<td>overall:</td>
<td>1095 ± 8</td>
<td>1.502 ± .007</td>
<td>32 5.0</td>
<td>15 2.3</td>
<td>46.9</td>
</tr>
</tbody>
</table>

Main effects:
corn
|                |                               |                                 | No. %            | No. %         |                                   |
|                | 1122 ± 10                     | 1.494 ± .009                    | 17 5.3           | 10 3.1        | 58.8                              |
| wheat          | 1067 ± 10                     | 1.510 ± .012                    | 15 4.7           | 5 1.6         | 33.3                              |
| significance:  | **                            | NS                              | NS NS            | NS NS         |                                   |

meat meal
|                |                               |                                 | No. %            | No. %         |                                   |
|                | 1097 ± 12                     | 1.490 ± .009                    | 19 5.9           | 6 1.9         | 31.6                              |
| SBM            | 1092 ± 10                     | 1.513 ± .011                    | 13 4.1           | 9 2.8         | 69.2                              |
| significance:  | NS                            | NS                              | NS NS            | NS NS         |                                   |

Data shown as mean ± SEM
* p<.05
** p<0.01
1 Percent of broilers housed (160/dietary treatment)
lower mean body weight.

By 40 days of age both grain type and protein source significantly affected total mortality and the incidence of SDS, but not SDS as a percent of total mortality. The broilers receiving the wheat based diets had significantly (p<.01) higher overall mortality and a higher incidence of SDS. Broilers fed diets which did not contain meat meal had significantly (p<.05) higher total mortality. There was no effect of cereal type or protein source on final body weights, but there was a significant cereal x protein interaction affecting feed conversion. This is shown graphically in figures 5.3 and 5.4. Adding meat meal to the diet decreased feed conversion ratios of broilers fed wheat based diets but increased the feed conversion ratios for broilers fed corn based diets.

5.4.3 Experiment 4: Since the 2 and 4 week results of Experiment 4 were similar, only the data from week 4 are presented (table 5.6) There was no significant effect of cereal type or protein source on total mortality or the incidence of SDS. SDS mortality as a percent of total mortality was significantly (p<.05) lower for the broilers fed diets which contained meat meal. Final body weights were significantly (p<.01) lower for the broilers fed the corn based diets but there was no effect of dietary protein source. There was no significant effect of cereal type or protein source on feed conversion.

5.5 DISCUSSION:

5.5.1 Effect of cereal type:
5.5.1.1 Mortality: In both floor pen studies (Experiments 2 and 3) total mortality and the incidence of SDS were higher at 40 days of age when wheat was substituted for corn in the diet of male broiler chickens. If the increase in total mortality was due solely to the increase in SDS mortality, SDS as a percent of total mortality would also be expected to increase. This was not the case since in neither floor study was SDS as a percent of total mortality affected by cereal type. There must have been, therefore, an increased incidence of death due to causes other than SDS associated with the wheat-based diets.

The increase in SDS mortality associated with the wheat based diets is in agreement with the work of Mollison (1983) who also found an increase in SDS mortality when broilers were fed wheat-soy versus corn-soy based diets. In a subsequent study, however, Mollison et al. (1984) found no effect of cereal type on the incidence of SDS.

In the brooder study (Experiment 4) there was no observed effect of cereal type on total mortality or the incidence of SDS. This may have been due to the shorter time period (4 versus 6 weeks) used in the study.

5.5.1.2 Mean body weight and feed conversion: In Experiments 2 and 4 the broilers fed the corn-based diets had significantly (p<.01) higher final body weights than the broilers fed the wheat based diets. This was unexpected since the diets were formulated to be isocaloric and isonitrogenous. Mollison et al. (1984) also obtained superior growth rates for corn-fed broilers. Sell et al. (1976) found that the inclusion
of fat in corn, oat and barley based diets of White Leghorns increased apparent ME, as measured experimentally, above that anticipated based on ME's calculated from reference values. This may explain the improved growth rate found for the corn-fed broilers. In Experiment 3, however, the same diets had no effect on final body weight.

Bowes et al. (1988) reported that a 25% feed restriction reduced SDS mortality and hypothesized that there was a relationship between growth rate and the incidence of SDS. In Experiment 2 the incidence of SDS was reduced by either a corn-based diet or the inclusion of meat meal. Both these factors increased growth rate as reflected in higher final body weights. Thus, the results of Experiment 2 do not support the hypothesis of Bowes et al. (1988).

In Experiment 2 and 3 there was a significant cereal x protein interaction affecting feed conversion. Feed conversion was the lowest with the wheat/meat meal diets and highest with the corn/meat meal diet. Thus the effect of meat meal on feed to gain ratios depends on the dietary cereal used. In Experiment 4 there was no effect of cereal type or protein source on overall feed conversion.

5.5.2 Effect of dietary protein source:

5.5.2.1 Mortality: In both floor pen studies (Experiments 2 and 3) the incidence of SDS was reduced when meat meal was included in the diet. In Experiment 3 there was also a decrease in total mortality with no effect on SDS as a percent of total mortality, indicating that death due to causes other than SDS was also reduced. In Experiment 2, however, SDS as a
percent of total mortality, but not total mortality, was decreased by inclusion of dietary meat meal. This would indicate that there was an increase in mortality due to causes other than SDS when meat meal was included in the diet. Thus the effect of dietary meat meal on the incidence of SDS was consistent in both floor pen studies but the effect on mortality due to causes other than SDS was not.

In the brooder study (Experiment 4) there was no effect of dietary meat meal on total mortality or on the incidence of SDS. SDS as a percent of total mortality, however, was significantly reduced. This effect was probably due to the nonsignificant decrease in SDS and the nonsignificant increase in total mortality associated with the inclusion of meat meal in the diet.

All of the diets used in this study were pelleted and contained varying quantities of soybean meal. The substitution of a portion of the soybean meal with meat meal appeared to reduce the incidence of SDS. Proudfoot et al. (1984) proposed that a toxic factor(s) was produced when protein supplements were subjected to pelleting which may be involved in causing SDS. If this were the case, it can be speculated that the toxic factor(s) must be produced when soybean meal, and not meat meal, is pelleted since reduction in the quantity of soybean meal in the diet reduced the incidence of SDS. It is more likely, however, that the inclusion of meat meal supplies a previously unidentified factor present in animal protein which provides some protection against the occurrence of SDS. One possibility is taurine. Taurine is a naturally occurring amino acid found
in all animal tissues but which is almost nonexistent in plants (Jacobsen and Smith, 1968). Taurine has been shown to be a membrane stabilizer (Kuriyama, 1980) and may be providing protection to the cardiac cell membranes and reducing the incidence of SDS.

5.5.2.2. Mean body weight and feed conversion: In Experiment 1 the inclusion of meat meal in the diet resulted in superior growth rates as reflected in significantly higher final body weight. In Experiments 2 and 3, however, there was no effect of dietary protein source on the incidence of SDS.
6.0 THE EFFECT OF A TAURINE TRANSPORT INHIBITOR ON THE INCIDENCE OF SUDDEN DEATH SYNDROME IN MALE BROILER CHICKENS

6.1 ABSTRACT:

Guandinoethyl sulfonate (GES), which has been found to inhibit taurine transport in rats, was added to the diet of male broiler chickens to study the effect on tissue taurine content and the incidence of Sudden Death Syndrome (SDS). GES was included in the diet at 0, .25 and .50% from one to two weeks of age. In the third week the levels were modified to 0, 1.50 and .50% respectively, and maintained at these levels through to the end of the fourth week.

The broilers receiving 1.50% GES supplementation during weeks 3 and 4 had significantly lower cardiac concentrations at 4 weeks of age, but significantly higher brain taurine concentrations than controls. At 4 weeks of age cardiac taurine levels of the 1.50% GES supplemented broilers were 85% of controls while brain taurine levels were 129% of controls. It appeared that GES supplementation at 1.50% prevented the normal increase in cardiac taurine concentration and decrease in brain taurine concentration that occurs during the development of the male broiler chicken. Although the 2 week brain taurine concentrations of the .25% GES supplemented broilers were significantly higher than controls there were no significant differences at 3 and 4 weeks of age.

Of the 360 broilers housed, only 4 of 8 deaths were attributed to SDS. Neither total nor SDS mortality was significantly affected by GES supplementation.
6.2 INTRODUCTION:

Sudden Death Syndrome (SDS) refers to a condition in which apparently healthy, fast-growing broiler chicks die suddenly from no apparent cause. Immediately preceding death there is a short, wing-beating convulsions so that the majority of affected broilers are found dead lying on their backs (Newberry et al., 1985; Bowes and Julian, 1986). As a result, the condition is often referred to as "Flip-Over disease". SDS has also been referred to as "Acute Death Syndrome", "heart attack", "lung edema" and "died in good condition" (Merck, 1986).

SDS is the main cause of mortality in Canadian broiler flocks with incidences in individual flocks varying from .71% to 4.01% of broilers housed (Riddell and Springer, 1985). Broilers of all ages are affected starting as early as two days of age and continuing through to marketing age with peak mortality usually occurring between 3 and 4 weeks of age. The condition appears to be influenced by the sex of the chicken since more than 70% of SDS affected broilers are male (Hemsley, 1965; Brigden and Riddell, 1975; Steele and Edgar, 1982; Riddell and Springer, 1985).

The actual cause of death in SDS cases is unknown. Some authors have found lung congestion and edema and have proposed that the lung congestion was a result of heart failure and that affected broilers die of suffocation (Brigden and Riddell, 1975; Ononiuwu et al., 1979b). Further studies have shown that lung congestion is not a consistent feature and that freshly dead broilers diagnosed as dying from SDS had no lung congestion (Riddel and Orr, 1980). This suggests that lung congestion is a
postmortem change probably related to position at death.

SDS death appears to involve heart damage. Microscopic examination of cardiac muscle from SDS affected broilers showed degeneration of the fibers, separation of the fibers by edema, and infiltration of heterophils (Ononiwu et al., 1979b). Julian and Bowes (1987) have suggested that the death is due to left ventricular fibrillation, but there is still no conclusive evidence as to the cause of this fibrillation. It has also been suggested that SDS is a metabolic disease related to high carbohydrate intake and that a build up of intermediary metabolites causes cardiac arrhythmias (Julian and Bowes 1987).

An earlier study in this laboratory showed that the incidence of SDS was influenced by the source of dietary protein. The inclusion of meat meal in the diet of male broiler chickens significantly reduced the incidence of SDS.

One chemical difference between animal and plant tissues is the presence of taurine in animal tissues. Taurine, or 2-aminoethane sulfonic acid, is a naturally occurring amino acid found in all vertebrate tissue, but which is almost non-existent in plants (Jacobsen and Smith, 1964; Kataoka and Onhishi, 1986).

Interest in taurine increased when it was discovered to be essential for cats. In this species, a deficiency of taurine results in retinal degeneration leading to blindness (Berson et al., 1976).

The concentration of taurine in heart tissues, relative to other tissues, is high in all animal species studied and may represent close to 50% of the total free amino acids present (Kocsis et al., 1976). The myocardial metabolism of rats has
been reported to be significantly altered by a depletion of cardiac taurine (Mozaffari et al., 1986). Taurine deficient cats have been shown to suffer from myocardial failure (Pion et al., 1987). Reduced cardiac taurine concentrations have been found to be associated with cardiomyopathies in turkeys (Schaffer et al., 1982). Furazolidone treated turkey poults showed an increase in the incidence of cardiomyopathies and had significantly reduced cardiac taurine concentrations. Administration of exogenous taurine to turkeys treated with furazolidone restored myocardial taurine to normal levels but did not alter the incidence of furazolidone induced cardiomyopathies.

Cardiac taurine is acquired either from the diet or from biosynthesis. In rats, a marked decrease in tissue taurine concentration has been achieved by supplementing with taurine transport inhibitors and thus preventing tissue uptake of taurine (Huxtable et al., 1981).

Guandinoethyl sulfonate (GES) is a structural analogue of taurine and in vitro inhibits taurine transport (Azari et al., 1979). Cardiac taurine concentrations of rats can be reduced in vivo to 20-30% of normal while the concentrations of the other amino acids remain unaffected (Huxtable et al., 1981). The irreducible portion is due to biosynthesis within the heart.

The effects of GES supplementation on tissue taurine concentrations vary between species (Huxtable and Lippincott, 1981). Mice are similar to rats in that taurine concentrations are reduced by a 1% GES supplementation in the drinking water. Cats are able to metabolize GES to taurine so that it does not
have an inhibitory effect. Guinea pigs are intermediate in their ability to metabolize GES.

Since GES administration is effective in reducing taurine concentrations in the tissues of mice and rats, both omnivores, it may be possible to use GES supplementation to reduce taurine concentrations in the tissues of the omnivorous chicken.

The purpose of this experiment was two fold. First to study the effect of GES supplementation on the tissue taurine content of male broiler chickens, and, second, to see if there was any effect on the incidence of SDS.

6.3 MATERIALS AND METHODS:

6.3.1 Design and Treatments: Three hundred and sixty day-old commercial male broiler chicks (Peterson X Arbor Acre) were reared in Peterson battery brooder cages to four weeks of age. The chicks were wing-banded and randomly distributed among 36 pens at density of 10 chicks/pen. The brooders were 68 x 99 cm in size giving a space allotment of .07 m^2/broiler. Chicks from nine of the pens were reserved for the supply of organ samples for taurine analysis while the chicks of the remaining twenty-seven pens were used for a mortality study. The mortality study used a completely randomized design with 9 replications per treatment.

For the first week all the broilers received a taurine free basal diet (table 6.1). From the second week to the end of the study, the broilers were divided into three treatment groups. Treatment A was a control and these broilers continued to receive the basal diet throughout the experiment. During week 2
Table 6.1 Composition and calculated analysis of the basal diet used in Experiment 5

<table>
<thead>
<tr>
<th>INGREDIENT</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wheat</td>
<td>27.9</td>
</tr>
<tr>
<td>Corn</td>
<td>27.9</td>
</tr>
<tr>
<td>Soybean meal (47% CP)</td>
<td>35.8</td>
</tr>
<tr>
<td>Corn oil</td>
<td>4.7</td>
</tr>
<tr>
<td>Limestone</td>
<td>1.7</td>
</tr>
<tr>
<td>Dicalcium phosphate (16% Ca; 20% P)</td>
<td>1.5</td>
</tr>
<tr>
<td>Premix</td>
<td>0.4</td>
</tr>
<tr>
<td>D,L-Methionine</td>
<td>0.2</td>
</tr>
</tbody>
</table>

Calculated analysis:
- Crude protein (%) 22.5
- Metabolizable energy (kcal/kg) 3100
- Calcium (%) 1.00
- Phosphorus, available (%) 0.45
- Methionine (%) 0.55
- Lysine (%) 1.32

1 Premix provided the following per kg diet: 9000 IU vitamin A; 1500 IU vitamin D3; 10 IU vitamin E; 0.5 mg vitamin K; 0.007 mg vitamin B12; 0.4 mg thiamin; 6 mg riboflavin; 1 mg folic acid; 0.15 mg biotin; 12 mg pantothenic acid; 35 mg niacin; 4 mg pyridoxine; 1000 mg choline; 2 gm iodized salt; 60 mg manganese; 5 mg copper; 50 mg zinc and 0.1 mg selenium.
broilers in Treatment B received the basal diet supplemented with .25% GES. This was increased the third week to 1.50% and remained at this level to the end of the study. The broilers of Treatment C received the basal diet supplemented with .50% GES from week 2 to the end of the study. Each dietary treatment was fed to twelve pens - nine for the mortality study and three for the provision of organ samples.

6.3.2 Management: The broilers received 23 hours of light per day and were provided feed and water ad libitum. Initial brooder temperature was set at 35°C and was reduced by 2.5°C weekly. At three weeks of age the brooder heaters were turned off.

The broilers were checked twice daily for mortality and all dead broilers necropsied. Death was attributed to SDS if there was no evidence of other disease and the broilers were in good body condition with a full digestive tract, a small or empty gall bladder and contracted ventricles.

At 2, 3 and 4 weeks of age mean body weight and feed consumption were determined for all broilers on a pen basis (36 pens). For the nine pens reserved for the supply of organ samples, two broilers per pen were randomly selected each week. They were weighed and killed by cervical dislocation and the hearts and brains removed. All samples were immediately frozen and stored at -20°C until analyzed for taurine content.

Sakaguchi et al. (1982) reported that the taurine concentration of fish muscle (Seriola quinquerediata) was not significantly altered by 40 days of ice storage. It was therefore assumed that the taurine concentrations of the chick heart and brain
samples would not be significantly affected by storage at -20°C.

6.3.3 Synthesis of GES: Guanidinoethyl sulfonate was synthesized according to the procedure of Huxtable et al. (1979). S-methylisothiourea (300 g) was mixed with taurine (180 g) dissolved in concentrated ammonium hydroxide (360 ml). The mixture was heated to 60°C and maintained at this temperature until the evolution of gas ceased (overnight). (WARNING: methane thiol, a toxic and foul smelling gas, was evolved in this reaction). The crystals formed were filtered and then recrystallized three times from water. Taurine contamination was detected by use of the amino acid analyzer. Residual taurine contamination was found to be .27%.

The S-methylisothiourea and taurine were purchased from Sigma (Sigma Chemical Co., St. Louis, Missouri) while the concentrated ammonium hydroxide was obtained from BDH (BDH Inc., Vancouver, BC).

6.3.4 Taurine analysis: Prior to analysis, the heart and brain samples were freeze-dried and ground in a mortar and pestle.

Heart samples were prepared for taurine analysis by an extraction technique. Approximately 75 mg of freeze-dried sample were homogenized in 2.5 ml of deionized water. Two ml of the homogenate were transferred to a centrifuge tube containing 8 ml of 10% (w/v) sulfosalicylic acid, mixed well and allowed to sit for 30 minutes. The mixture was then centrifuged at 10000xg for 6 minutes and 5 ml of the supernatant transferred to a 10 ml volumetric flask. The flask was filled to volume with 0.2M sodium citrate buffer (pH 2.2), mixed well and allowed to sit
overnight in the refrigerator before analysis.

Brain samples prepared using the above extraction procedure contained a substance which eluted closely with taurine. It was therefore necessary to prepare the brain samples for taurine analysis using an acid hydrolysis procedure. The substance was destroyed and clear separation of taurine was possible.

Approximately 75mg of freeze-dried brain sample was placed in a flat-bottom boiling flask. After the addition of approximately 20ml of 3N HCl the samples were autoclaved at 121°C and 15 psi for 17 hours. HCl was removed by rotoevaporation in a water bath at 60°C. Each sample was redissolved in 0.2M sodium citrate buffer (pH 2.2), filtered through a medium porosity gooch apparatus and transferred quantitatively into a 50 ml volumetric flask containing 3 ml 0.1N NaOH. The flask was filled to volume with the sodium citrate buffer and stored overnight in the refrigerator before analysis.

Taurine content was measured on a Beckman 6300 automatic amino acid analyzer. A sequence of two temperatures (50°C and 65°C), three buffers (Na-A, Na-B and Na-C in order of increasing pH) and a cation exchange column were used. Sodium hydroxide was used to regenerate the column after each sample. A buffer flow rate of 15 ml/h and a solvent flow rate of 8 ml/h was used. Recovery of taurine was close to 100%.

6.3.5 Statistical analysis: Performance and mortality data, as well as taurine content, were statistically analyzed as a completely random design using one-way analysis of variance procedures of the Statistical Analysis System (SAS Institute
Inc., 1982). Any significant differences were further analyzed using the Ryan-Einot-Gabriel-Welsch multiple range test. An arcsin transformation was used before analyzing the mortality data.

6.4 RESULTS:

Final mean body weights, feed consumption and feed conversion data from Experiment 5 are shown in table 6.2. GES supplementation had no significant effect on final body weight, but did significantly (p<.05) increase feed consumption. This is reflected in a significantly (p<.05) higher feed to gain ratio.

Cardiac (table 6.3 and figure 6.1) and brain (table 6.4 and figure 6.2) taurine concentrations were affected by GES supplementation. At 4 weeks of age the broilers receiving 1.50% GES dietary supplementation had cardiac taurine concentrations that were significantly (p<.05) lower than controls. At 3 and 4 weeks of age the broilers receiving 1.50% GES supplementation had brain taurine concentrations that were significantly (p<.05) higher than those of the controls.

Cardiac taurine concentrations of the control broilers and the broilers receiving .50% GES supplementation increased over time so that at 4 weeks of age the concentrations were significantly higher than at 1 week of age. This was not the case for the broilers of Treatment B. The broilers in Treatment B received .25% GES supplementation during week 2 and 1.50% GES thereafter. While on the .25% supplementation, cardiac taurine concentrations increased so that the concentration at 2 weeks of
Table 6.2 Effect of GES supplementation on mean body weight, feed consumption and feed conversion (Experiment 5): Results at 4 weeks of age

<table>
<thead>
<tr>
<th>DIET</th>
<th>FINAL BODY WEIGHT (g/broiler)</th>
<th>FEED CONSUMPTION (g/broiler)</th>
<th>FEED CONVERSION (g feed/g gain)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>$915 \pm 11^a$</td>
<td>$1376 \pm 23^a$</td>
<td>$1.580 \pm 0.031^a$</td>
</tr>
<tr>
<td>B</td>
<td>$907 \pm 9^a$</td>
<td>$1430 \pm 16^ab$</td>
<td>$1.656 \pm 0.014^b$</td>
</tr>
<tr>
<td>C</td>
<td>$927 \pm 12^a$</td>
<td>$1454 \pm 18^b$</td>
<td>$1.647 \pm 0.010^b$</td>
</tr>
</tbody>
</table>

Data shown as mean $\pm$ SEM

1 Diets all had 0% GES week 1. Week 2, diet A had 0%, diet B 0.25% and diet C 0.50%. Weeks 3 and 4, diet A had 0.0%, diet B 1.50% and diet C 0.50%

a,b Means within a column with different superscripts are significantly different (p<.05)
Table 6.3 Effect of GES supplementation on cardiac taurine content (umol/g wet weight) (Experiment 5)

<table>
<thead>
<tr>
<th>DIET</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>23.81 ± 0.47&lt;sup&gt;ax&lt;/sup&gt;</td>
<td>28.34 ± 0.68&lt;sup&gt;ay&lt;/sup&gt;</td>
<td>27.60 ± 0.58&lt;sup&gt;ay&lt;/sup&gt;</td>
<td>31.64 ± 1.23&lt;sup&gt;az&lt;/sup&gt;</td>
</tr>
<tr>
<td>B</td>
<td>22.66 ± 1.46&lt;sup&gt;ax&lt;/sup&gt;</td>
<td>28.14 ± 1.21&lt;sup&gt;ay&lt;/sup&gt;</td>
<td>28.11 ± 0.91&lt;sup&gt;ay&lt;/sup&gt;</td>
<td>26.90 ± 1.41&lt;sup&gt;bxy&lt;/sup&gt;</td>
</tr>
<tr>
<td>C</td>
<td>24.55 ± 0.48&lt;sup&gt;ax&lt;/sup&gt;</td>
<td>29.18 ± 0.87&lt;sup&gt;ayz&lt;/sup&gt;</td>
<td>25.23 ± 2.16&lt;sup&gt;axy&lt;/sup&gt;</td>
<td>30.37 ± 0.67&lt;sup&gt;az&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Data shown as mean ± SEM of 6 animals per point

1 Diets all had 0% GES week 1. Week 2, diet A had 0%, diet B 0.25%, and diet C 0.50%. Weeks 3 and 4, diet A had 0%, diet B 1.50% and diet C 0.50%

a, b Means within a column with different superscripts are significantly different (p<.05)

x, y, z Means within a row with different superscripts are significantly different (p<.05)
Figure 6.1 Effect of GES supplementation on cardiac taurine concentration (Experiment 5)

Age In weeks

GES (% diet)

- CONTROL
- 0/.26/1.50
- 0/.50/50

- GES levels at wk1/wk2/wk3,4

a,b means within a week with different letters are significantly different.
Table 6.4 Effect of GES supplementation on brain taurine content (umol/g wet weight) (Experiment 5)

<table>
<thead>
<tr>
<th>DIET</th>
<th>WEEKS OF AGE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
</tr>
<tr>
<td>A</td>
<td>5.88 ± .30\text{a}x</td>
</tr>
<tr>
<td>B</td>
<td>5.69 ± .16\text{axy}</td>
</tr>
<tr>
<td>C</td>
<td>5.88 ± .10\text{axy}</td>
</tr>
</tbody>
</table>

Data shown as mean ± SEM of 6 animals per point

1 Diets all had 0% GES week 1. Week 2, diet A had 0%, diet B .25% and diet C .50%. Weeks 3 and 4, diet A had 0%, diet B 1.50% and diet C .50%

a,b Means within a column, with different superscripts are significantly different (p<.05)

x,y,z Means within a row, with different superscripts are significantly different (p<.05)
Figure 6.2 Effect of GES supplementation on brain taurine concentration (Experiment 5)

![Bar chart showing the effect of GES supplementation on brain taurine concentration.](chart.png)

- **GES (% diet):**
  - CONTROL
  - 0/.25/1.50
  - 0/.50/.50

- **Age in weeks:**
  - Week 1
  - Week 2
  - Week 3
  - Week 4

- **Taurine concentration (μmol/g wet wt):**

- **a, b means within a week with different letters are significantly different.**

- GES levels at wk1/wk2/wk3-4
age was significantly higher than at 1 week of age. By 4 weeks of age, however, the cardiac taurine concentrations were no longer significantly different from those at 1 week of age.

Brain taurine concentration of the broilers in the control group and the broilers receiving .50% GES supplementation decreased over time so that at 4 weeks of age taurine concentrations were significantly lower than at 1 week of age. This was not the case for the broilers of Treatment B which received .25% GES supplementation during week 2 and 1.50% GES thereafter. Brain taurine concentrations were maintained so that the concentration at 4 weeks of age was not significantly different from the concentration at week 1.

In the mortality study portion of the experiment only six broilers died. Treatments A, B and C had 1, 2 and 3 broilers die respectively. Each treatment had only 1 broiler die of SDS.

6.5 DISCUSSION:

From this study it was concluded that GES supplementation affects tissue taurine content in male broiler chickens quite differently from that of rats (Huxtable et al., 1981). In rats taurine concentrations were reduced in all tissues examined, by a 1% supplementation of GES in the drinking water. In this experiment, supplementation with 1.50% GES in the feed resulted in 4 week cardiac taurine levels that were 85% of controls, but brain taurine levels that were 129% of controls.

The cardiac taurine concentrations of the control broilers increased over time. It would appear that GES supplementation prevented this increase rather than causing a reduction in
taurine concentrations to occur. The brain taurine concentra-
tions of the control broilers decreased over time. GES
supplementation appeared to prevent this decrease.

Brain taurine concentrations vary between species, but in
most, taurine concentration is highest at birth (Sturman et al.,
1977). While the concentration of most of the amino acids in
the brain either increase or remain constant over time, the
concentration of taurine decreases with age. It would appear
that taurine concentrations in the brain of male broiler
chickens follows this pattern of decreasing with age.

Although an animal's brain taurine concentrations are
highest at birth, their biosynthetic capacity is at its lowest
at this time. It has been suggested that the brain has an
efficient and highly selective transport system for attaining
and maintaining taurine concentrations (Sturman et al., 1977).
It is possible that the brain was able to compensate for the
effect of GES inhibition by increasing the half-life of the
endogenous taurine. This would explain the apparently higher
levels of brain taurine in the 1.50% GES supplemented broilers.

Cardiac taurine levels also change during development but
the direction of change varies between species (Sturman et al.,
1977). In some species, such as the rat and rabbit, the
concentrations fluctuate during development but there is little
difference between concentrations at birth and at maturity. In
other species, such as the cat and monkey, there is no apparent
change in cardiac taurine concentration during development.
Cardiac taurine levels of the male broiler chicken appear to
increase during development.
GES supplementation did not affect total mortality or the incidence of SDS. Since cardiac taurine levels were only reduced to 85% of controls it is premature to rule out a role for taurine in the prevention of SDS in broilers.
7.0 CONCLUSIONS:

Four factors were studied for possible involvement in the etiology of SDS in male broiler chickens—lactic acid, cereal type, protein source and taurine. It appears unlikely that lactic acid is involved in SDS since supplementation with calcium lactate to 7.5% of the diet had no effect on the incidence of SDS. Feeding glucose based diets did not modify the effect of lactic acid supplementation.

Cereal type was shown to affect the incidence of SDS. Male broiler chickens raised on wheat-based diets had a higher incidence of SDS than those raised on corn-based diets. This increase in SDS mortality could not be explained on the basis of growth rate since the effect of cereal type on mean body weight was inconsistent across the three studies.

Protein source was also shown to effect the incidence of SDS. The inclusion of meat meal in the diets of male broiler chickens reduced the incidence of SDS. It was suggested that taurine present in meat meal, but absent in soybean meal, was involved in protecting the broiler from SDS. This hypothesis was tested by reducing cardiac taurine levels with the aid of a taurine transport inhibitor, guanidinoethyl sulfonate (GES). At 4 weeks of age, the broilers receiving 1.5% GES supplementation in the feed had cardiac taurine concentrations that were 85% of controls and there was no effect on the incidence of SDS. It is possible that a greater depletion of taurine is required before an effect can be seen, but it seems unlikely that the presence of taurine in meat meal is the reason for the increased protection against SDS.
The effect of GES supplementation on broiler tissue taurine concentration merits further investigation. While the 4 week cardiac taurine concentrations of the 1.5% GES treated broilers were 85% of controls, the brain levels were 129%. It appeared that GES was preventing the normal increase in cardiac taurine concentrations and decrease in brain taurine concentrations that occur in the developing male broiler.

Bowes et al. (1988) reported that a 25% feed restriction reduced SDS mortality and hypothesized that there was a relationship between growth rate and the incidence of SDS. In Experiment 2 the incidence of SDS was higher when the broilers were fed a wheat-based rather than a corn-based diet or when meat meal was excluded in the diet. Both these factors reduced growth rate as reflected in significantly lower final body weights. The level of reduction in either case was less than the 41% reduction in body weight obtained by Bowes et al. (1988).

In Experiment 2, substituting wheat for corn resulted in only a 5% reduction in 40 day mean body weight. Excluding meat meal from the diet resulted in only a 2% reduction in 40 day mean body weight. Mollison et al. (1984) reported that a 15% reduction obtained by using 13% feed restriction did not significantly affect the incidence of SDS. A greater reduction in growth rate may have been necessary to see an effect. It is, therefore, not possible to rule out the role of growth rate in the etiology of SDS on the basis of the results of Experiment 2.

In Experiment 1 growth rate was also reduced by dietary treatment without an effect on the incidence of SDS. There was
a 33% difference between the highest and lowest 4 week mean body weights obtained, but the incidence of SDS was not significantly affected. On the basis of the results of Experiment 1, it appears unlikely that there is a relationship between growth rate and the incidence of SDS.
8.0 LITERATURE CITED:


