THE EFFECT OF FEED PROCESSING AND FEED TEXTURE ON BODYWEIGHT, FEED CONVERSION AND MORTALITY IN MALE BROILERS

by

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CHAPTER I

1. INTRODUCTION

A number of specialised poultry breeding companies have been in existence since the late 1940's to early 1950's, breeding chickens for meat consumption. Over the period 1957 to 1991, the age to slaughter and the amount of feed required to produce a given quantity of chicken meat has been more than halved (Havenstein, Ferket, Scheideler and Larson, 1994). The bodyweight of commercial broilers (the average of both sexes) has increased from about 2000 g at 56 days in 1976 to about 3100 g in 1991. Thus, 56 day broiler bodyweight has increased over that 15 year period by approximately 1100g (73g / year).

This increase has effectively reduced marketing age to a given bodyweight by about 1day/year from 1976 to 1991 (Havenstein *et al*., 1994). The main increase in growth rate is manifested primarily in the first four weeks after hatching (Marks, 1979).

The very fast growth rate in modern broiler strains has been due to constant genetic selection and improvement in nutrition (Portsmouth and Hand, 1987). Sherwood (1977) and Havenstein *et al*. (1994) indicated that most of the increase in bodyweight, probably 85 to 90 %, has come about due to genetic selection for increased bodyweight applied by commercial broiler breeder companies. Havenstein *et al*. (1994) also compared a 1991 diet with a 1957 diet. The 1991 diet increased the bodyweight of the Athens – Canadian Randombred Control (ACRBC) broiler by 20 to 26% for the different

age and sex groups over that observed with the 1957 diet. For the Arbor Acres (AA) broiler, the percentage advantage for the 1991 diet over the 1957 diet was similar (18 to 26%) to that observed with the ACRBC at 42 days of age.

However, not only did growth rates increase over the last 40 years, but also mortality rates of broilers. The average mortality rate for the 1991 AA birds at 42 days of age (9,1 %) was almost three times that of the ACRBC birds (3,3%). Most of the mortalities for the AA birds after 21 days of age were associated with flip-overs, ascites and leg problems. Mortality rates in the AA birds were consistently higher in males than in females (Havenstein *et al*.,1994).

Havenstein *et al.* (1994) found that the feed conversion at 42 days for the ACRBC birds on the 1991 diet was 2,51 vs 2,04 for the AA birds on the same diet. This is an improvement of 23%. The geneticists thus succeeded in improving growth and feed conversion, but at the expense of liveability, especially when looking at farms ∃900m above sea level (Buys, 1996).

Buys (1996) reported that three organ systems of the modern broiler have become its Achilles heel and have not improved at the same pace as the growth rate. They are the respiratory (cardiovascular) system, skeletal system and immune system.

Constant genetic selection and improvement in nutrition have led to a very fast growth rate in modern broiler strains (Zubair and Leeson, 1996). Unfortunately, early fast growth rate in broiler chickens is accompanied by a number of problems – namely, increased body fat deposition, a high incidence of metabolic diseases, high mortality and a high incidence of skeletal disease (Leeson and Summers, 1988).

Growth management is necessary to assist in achieving optimum performance in terms of health, welfare, liveweight, uniformity, feed conversion and meat yield. Growth is managed in the first 14 days to less than maximum daily weight gain in a manner designed to optimise early physiological development and in particular, the development of the heart and lungs, immune and skeletal systems. The two principal methods of regulation involve nutritional (i.e. control of feed and nutrient intake) and lighting programmes (Ross Breeders Limited, 1996).

Zubair *et al.* (1996) summarised the following methods of nutrient restriction; (a) physical feed restriction, (b) diet dilution, (c) chemical methods of feed restriction and (d) the use of low protein or low energy diets. Such a period of undernutrition is usually followed by compensatory growth. The effect of feed texture was discussed by Proudfoot and Hulan (1982a). They showed that birds on a crumbled starter and pelleted finisher diet had superior bodyweights if compared to birds on a all mash starter and finisher diet.

Blair, Newberry and Gardiner (1993) suggested that an increasing light pattern is beneficial in broiler flocks in reducing overall mortality, sudden death syndrome (SDS) mortality, the incidence of leg deformities and improving feed efficiency, with no reduction in bodyweight at market age. Gordon (1994) confirmed these results and reported that metabolism may be modified by providing an extended dark period, possibly due to changes in melatonin synthesis. Some of the physiological benefits of providing an extended dark period, such as reduced stress and improved immunoresponsiveness, may also occur during the early growth stages.

The objective of this study was to evaluate the influence of the feed manufacturing process and feed texture (crumbles and pellets, ground crumbles and pellets, and mash) on bodyweight, feed conversion and mortality, in male Ross 788 broilers, reared on the South African highveld at 1517 metres above sea level.

CHAPTER II

2. LITERATURE REVIEW

Metabolic diseases have become an important problem to the poultry industry. Genetic growth potential and high feed consumption have increased metabolism beyond the physiologic ability of the heart, lungs, liver and skeleton to perform normally and to keep growth and the body systems in balance. Diseases like flip-over or SDS, pulmonary hypertension syndrome (PHS) and musculoskeletal defects, are responsible for enormous economic losses to the poultry industry. The loss associated with these conditions is greater than that of infectious diseases (Julian, 1994).

Chambers, Gavora and Fortin (1981) estimated the change in carcass weight over the 20 year period from 1958 to 1978 using the Ottawa Meat Control bird. They reported that the 1978 broiler was approximately 2.3 times the size of the control at 47 days of age and had a greater proportion of fat.

Sherwood (1977) used the ACRBC broiler and the calendar year 1976 Hubbard broiler and placed the ACRBC broiler on the 1953 and the Hubbard broiler on the 1976 diets. At 56 days of age, the Hubbard broilers were 2.2 times as heavy as the ACRBC broilers. Fifteen years later, Havenstein *et al*. (1994) reported that the body-weight of the AA broiler on the 1991 diet was 3.9 times heavier than the ACRBC broiler on the 1957 diet. They further reasoned that the bodyweight of commercial broilers (the average of both sexes) had increased from about 2000g at 56 days in 1976 to about 3100g in 1991.

Ascites

Pulmonary hypertension (PH) caused by increased blood flow or increased resistance to flow in the lung results in right ventricular hypertrophy (RVH), valvular insufficiency, increased venous pressure and ascites (Julian, 1993).

Ascites secondary to right ventricular failure (RVF) occurs worldwide in growing broiler chickens and is a significant cause of mortality in many flocks (Riddell, 1991). The sudden increase in PHS in meat-type chickens in the 1980's was associated with a rapid increase in growth rate and feed conversion, as well as a more dense, high caloric, pelleted food, that supplied all the nutrients required for rapid growth and encouraged a high nutrient intake. The small stature of the modern meat-type chicken, the large heavy breast mass, the pressure from abdominal contents on air sacs, and the small lung volume compared to bodyweight, may all be involved in the increased incidence of PHS (Julian, 1993).

There is limited space for blood flow in the avian lung (Julian, 1993). The lung volume of the broiler as compared to the wild Red Jungle fowl is only 76 % on the basis of volume to body mass. As the broiler gets older, the lung volume to the body mass ratio worsens (Vidyadaran, King and Kassim, 1990). The small capillaries of the birds can expand only very little. This together with increased blood-flow because of rapid growth and the increased blood viscosity caused by polycythaemia because of hypoxia-induced hypoxaemia plays an important role in the increase of PHS (Julian, 1993). Julian (1993) reported that the increase of PHS at low and high altitude is related to the high oxygen requirement of rapid growth and the inability of the heart and lung to deliver sufficient oxygen to the tissue.

Research on meat-type chickens indicates that fast-growing broilers have a lower percentage haemoglobin oxygen saturation than slow-growing broilers (Julian and Mirsalimi, 1992). Another complicating factor is that the right atrioventricular valve is composed of a muscle flap made up mainly of muscle fibres from the right ventricle wall. The anatomy of the valve makes the bird very susceptible to valvular insufficiency (Julian, Friars, French and Quinton, 1987).

At necropsy there can be a varying quantity of clear yellow fluid and clots of fibrin in the abdomen. The liver may be swollen and congested, or firm and irregular with oedema and have fibrin adherent to the surface, or it may appear white, nodular and shrunken, with oedema and fibrosis under the capsule. There is mild to marked hydropericardium and occasionally there is pericarditis with adhesions. There is right ventricular dilation and hypertrophy of the right ventricular wall. The right auricle and vena cava are very dilated. Frequently there is thinning of the left ventricle. The lungs are extremely congested and oedematous (Jordan, 1990).

Not all broilers that die from RVF have ascites. Death may occur before clinical signs are observed and affected broilers frequently die on their back. At necropsy there may be a swollen liver, venous congestion, a dilated right atrium and vena cava,right ventricular hypertrophy (RVH), as well as marked lung congestion and oedema. Death is probably from respiratory failure. The intestine may or may not be empty, but the heart lesion will differentiate RVF from flip-over disease (Jordan, 1990).

Ascites was first reported in flocks reared at high altitudes in Bolivia (Hall and Machicao, 1968). The first report of ascites in South Africa

was by Buys and Barnes (1981). They reported an incidence of 2 to 12 percent up to the age of 56 days. The incidence of ascites is above 1% in some broiler flocks and many roaster flocks, and it is occasionally 15 - 20 % in roaster flocks (Julian *et al*., 1987).

There are many causes of ascites, which have been discussed by Julian (1993) and Huchzermeyer (1989). Julian *et al*. (1987) reported that rapidly growing broilers are more susceptible to increased pulmonary arterial pressure (resulting in right ventricular hypertrophy, right ventricular failure, and ascites) than slower-growing broilers.

Julian (1993) mentioned that incomplete oxygen saturation of haemoglobin is the most important cause of pulmonary hypertension (PH) at high altitude. Hypoxaemia causes polycythaemia by stimulating erythropoietin production. Polycythaemia increases blood viscosity and markedly increases the resistance to blood flow through the lung.

Huchzermeyer, de Ruyck and van Ark (1988) concluded that differences in the susceptibility to ascites in different commercial broiler strains can only be explained as genetic. The practical solution to the broiler ascites problem therefore rests in the selection for resistance to ascites in broiler breeding stock.

Huchzermeyer, van der Colf and Guinane (1989) reported that in an already marginal situation (e.g. moderate altitude), the increased oxygen (O_2) demand caused by cold, can produce an hypoxic state leading to pulmonary hypertension and ascites, or could contribute to an aggravation of the condition.

The use of pelleted feeds appears to be a contributing factor in the development of ascites, even when broilers are grown at low altitude. It is believed that any factor which increases chick growth or improves feed conversion also increases ascites (Lamas da Silva, Dale and Luchesi, 1988).

Sudden Death Syndrome

Sudden death syndrome (SDS) has also been described as acute death syndrome (ADS), heart attack and flip-over. It was first described as "oedema of the lungs" in 1965, and subsequently as "died in good condition" in 1972. The incidence reported in 1965 varied from 0,2 - 1,0 % and has since appeared to increase. In 1985 an average incidence of 1,95% with variation between flocks from 0,7 to 4,07% was reported in Saskatchewan (Riddell, 1993). Gardiner, Hunt and Newberry (1988) reported a mortality of 1,31 to 9,62% due to SDS.

Figures on incidence of SDS should be interpreted with caution. The non-specific lesions on which the diagnosis is based may introduce error. Some early reports may have included birds dying from suffocation due to panic in the barn under SDS (Riddell, 1993).

There are no specific lesions, but the supine position is significant when present and broilers that die from flip-over are invariably well fleshed and usually have ingesta in the crop and gizzard. The abdomen is mildly distended because affected broilers are fat and the intestine is dilated and filled with digesta and mucus. In young broilers the liver is large and frequently pale and fatty and the gallbladder is small or empty. The heart is bullet shaped with contracted ventricles and dilated, blood filled atria. The lungs are

congested and frequently very oedematous (although the oedema increases with time after death and is not prominent in broilers examined within a few minutes of death). All organs are moderately to severely congested. Small haemorrhages may be present on the liver and kidney. The bursa is large and normal, which again suggests that the bird was healthy immediately prior to death (Jordan, 1990).

Gardiner *et al*. (1988) remarked that the first deaths diagnosed as being due to SDS occurred on day 2 and the mortality rate reached a maximum when birds were between 21 and 27 days of age. After this plateau it decreased gradually over the remaining time. Jordan (1990) mentioned that mortalities can start within 72 hours of hatching and may continue up to 12 weeks of age, with peak mortality from 1 to 3 weeks, coinciding with the age at which feed conversion is best. However, flocks in which growth has been restricted during the first 3 weeks may experience higher than normal losses from SDS after 3 to 4 weeks, when compensatory growth take place. Seventy to seventy five percent of birds dying from SDS are males (Jordan, 1990; Riddell, 1993).

SDS is seen in apparently healthy, fast growing broilers that die suddenly with a short, wing-beating convulsive attack. Most affected broilers die on their back (Jordan, 1990). It is probably a metabolic disease in which an imbalance of metabolites or electrolytes results in ventricular fibrillation (Jordan, 1990). Greenlees, Eyre, Lee and Larsen (1989) demonstrated an increased myocardial irritability in fast growing compared to slower growing male broiler chickens at 3 weeks of age, which was not observed in 6 week old birds. They suggested that the increased myocardial sensitivity is due to rapid biochemical or endocrine changes, so that some birds are predisposed to SDS under certain conditions.

SDS is a problem of broiler chickens and the incidence is influenced by genotype. In an early report, a marked difference in incidence from 0,2 to 0,3% in less developed strains of broilers, to 0,6 to 1,0% in more developed White Rock strains were noted (Riddell, 1993). Most modern broiler strains of chickens are susceptible, but hereditability of SDS is low (Chambers, 1986).

In a study of the incidence of SDS in 23 experiments, the death rate from SDS showed a significant increase as the final flock bodyweight increased (Gardiner *et al*.,1988), but this correlation was not found in a study of 51 commercial flocks (Riddell and Springer, 1985). Ononiwu, Thomson, Carlson and Julian (1979) found that there is some difference between average weight of broilers dying of SDS and the flock average weight. The difference in favour of the SDS birds amounted to 2,34% at two weeks and 0,79% at four weeks.

Nutrition may also influence the incidence of SDS (Riddell, 1993). Proudfoot, Hulan and McRae (1982b) found that mortality due to SDS was significantly higher for birds fed a crumble-pellet diet in its usual form or in a ground form, compared with those fed an all-mash diet. They concluded that the higher incidence of SDS was due to some factor(s) in the pelleting process itself, rather than the rapid growth resulting from the higher density of pelleted feeds.

Skeletal Disorders

Thorp (1994) reviewed skeletal disorders in the fowl. He reported that selection pressure for production traits in modern lines of poultry has placed increasing demands on skeletal integrity.

Disruption of the normal process of skeletal growth and homeostasis results in bone diseases that are manifested throughout the modern poultry industry. The major developmental or metabolic skeletal disorders of meat-type birds are valgus-varus deformation (VVD) of the intertarsal joint and tibial dyschondroplasia (TD) (Riddell, 1993). The factors that influence the incidence of VVD and TD include growth rate, weight gain, behavioural characteristics, strain and housing, as discussed by Thorp (1994).

VVD of the intertarsal joint is characterised by outward or inward angulation of the distal tibiotarsus, with similar but less severe angulation in the proximal tarsometatarsus (Riddell, 1993). Abnormal limb posture can also originate from the femur. Slight intertarsal valgus of less than 10° is physiological, valgus deformity greater than 20° or varus deformity is abnormal (Thorp, 1994). The prevalence varies from 0,5 to 2,0% in normal broiler flocks, but occasionally affects 5 - 25% of male broilers in problem flocks (Julian, 1984).

Broilers may be affected with VVD before one week of age and the prevalence increases with birds becoming affected throughout the life of a flock (Riddell *et al*.,1985). Approximately 70% of affected birds are males. The defect may affect both legs, but is often unilateral with the right leg more commonly affected than the left leg (Riddell *et al*., 1985). This indicates that limb dominance exists in the fowl (Thorp, 1994).

VVD is related to over-nutrition and rapid growth. It may be due to uneven growth of the two tarsal bones, or the growth plate at the end of the distal tibia, or asymmetrical tendon tension on the fast growing bones (Julian, 1994). Thorp (1994) reported that altered load bearing and functional activity with more rapid growth, may contribute to a failure in the mechanisms that maintain torsion within limits. Lilburn (1994) suggested that in modern commercial broilers, the femur may be the weak link in the increased bodyweight : skeletal weakness equation. He also reported that the first 7 days are critical to overall skeletal development.

TD is characterised by an abnormal mass of cartilage in the metaphysis of the proximal tibiotarsus. A 30% incidence of TD is common in broiler chicken and turkey flocks (Riddell, 1993). Mild to severe TD is present in the proximal tibia in many fast-growing male broilers, but it does not cause lameness unless the diaphysis is weakened enough for compression to occur (Julian, 1994).

Julian (1994) reported that fast-growing broilers frequently show a mild to moderate painful lameness after three to four weeks of age. The birds hobble and walk as if leg movement and weight-bearing were painful. Most of these broilers have no deformity or lesion that would explain their behaviour. This lameness is associated with rapid growth and made worse by diets low in calcium or high in magnesium.

Immune Performance

The genetic make-up of commercial broilers has been dramatically changed by selection for production characteristics. However, such

selection criteria may result in a negative genetic relationship between the production and immune endpoints (Van der Zijpp, 1983). Qureshi and Havenstein (1994) compared the immune response of a 1957 male broiler (ACRBC) to a 1991 male broiler (AA). The 1957 males fed 1957 diets had the highest total IgM and IgG anti-sheep red blood cell antibodies. It showed that genetic selection towards enhanced performance traits has negatively influenced humoral immunity, with little or no effect on cellular immunity.

Qureshi and Miller (1991) concluded that different broiler genetic lines exhibit differences in their macrophage function potential.

Ross 788 Broiler

The Ross 788 broiler was developed in South Africa by Ross Poultry Breeders (Pty) Ltd. They reported that over the years, the continued selection for resistance to ascites and performance on the South African Highveld has resulted in a bird with extremely low mortality under these conditions. Excellent performance is achieved without sacrificing growth rate, feed conversion efficiencies, meat yield or conformation. The broiler has a proven track record under extreme conditions of environment (Ross Poultry Breeders (Pty) Ltd, 1994).

Feed Pelleting

Early feeding trials suggested that pelleting improved both growth and feed efficiency (Heywang and Morgan, 1944; Arscott, Hulit and Poutz, 1957). These early observations were later confirmed in several bird species such as the chicken (Hussar and Robblee,

1962), turkey (Blakely, MacGregor and Hanel, 1963) and Japanese quail (Angulo, Bureau, Miquel and Esteve-Garcia, 1993). Despite this evidence, Bayley, Summers and Slinger (1968) have been unable to detect any beneficial responses of growth and feed efficiency due to pellet feeding. The variation in observations may be due to : 1) different quality of pellets used in the different studies, or the use of different production procedures; 2) use of different species and different ages; 3) comparison of the effect of pelleting in feeds with variable energy levels due to differences in fat supplementation and fiber-containing ingredients (Plavnik, Wax, Sklan and Hurwitz, 1997).

Several mechanisms have been suggested to explain the effects of pelleting on performance variables :

- The decrease in diet volume and the large particle size were considered to facilitate feed or energy consumption and hence promote growth (Plavnik *et al*., 1997). The elimination of the growth effect when the pellets were ground (Arscott *et al*., 1957) may be taken as supporting this hypothesis.
- Growth promotion by itself may be sufficient to explain improvement in feed efficiency by pelleting (Plavnik *et al*., 1997).
- Reduced energy expenditure in the process of feed consumption itself (Jensen and Falen, 1973).
- Inactivation of heat-labile toxic factors in feeds (Alfred, Fry, Jensen and McGinnis, 1957).
- Improvement in digestion or utilization of feed ingredients, especially those high in fiber (Summers, Bentley and Slinger, 1968).
- Chemical changes in dietary carbohydrates resulting in increased metabolizable energy values (Summers *et al*., 1968). Although the improvement in metabolizable energy by pelleting has not been substantiated (Sibbald, 1977).

- Increases in protein/amino acids biovailability (Moran and Summers, 1970).

There are conflicting reports in the literature on some aspects of metabolic diseases. Julian *et al*. (1987) suggested that rapidly growing broilers are more susceptible to increased pulmonary arterial pressure resulting in right ventricular hypertrophy, right ventricular failure and ascites than slower-growing broilers. However, Dale and Villacres (1988) have reported that rapid early growth was not a factor influencing the incidence of ascites.

The death rate from SDS showed a significant increase as final flock bodyweight increased (Gardiner *et al*.,1988), but this correlation was not found by Riddell *et al*. (1985). Ononiwu *et al*. (1979) found that the average weight of birds dying from SDS, were heavier than the flock average at two, three and four weeks of age. It was concluded by Proudfoot *et al*. (1982b) that the higher incidence of SDS was due to some factor(s) in the pelleting process itself, rather than rapid growth resulting from the higher density of pelleted feeds.

Broilers that consume pelleted feed have frequently been shown to have higher incidences of ascites than broilers that consume the same diet in mash form. This is true for high and low altitudes (Lamas da Silva *et al*.,1988). Lamas da Silva *et al*. (1988) did not compare the effect of ground crumbles and pellets with normal crumbles and pellets. It is therefore not known what the effect of the pelleting process on ascites is.

CHAPTER III

3. MATERIALS AND METHODS

3.1. MODEL SYSTEM AND JUSTIFICATION OF THE MODEL

Six thousand day-old Ross 788 male broiler chickens were selected by systematic random sampling, originating from a specific broiler breeder flock (37 weeks old). The chickens were hatched from the same setter and hatcher.

The broilers were kept in a controlled environmental house. It provided the birds with a hygienic and protected environment which was free from intrusion by predators and in which extremes of temperature and humidity were prevented. Feed and water was readily accessible to individual birds.

For this research, broilers could not be replaced by other species. The Ross 788 was used because it is the broiler currently used by Earlybird Farm (Pty) Ltd. The Ross 788 is known for its resistance to ascites and performance on the Highveld. Only male broilers were used to exclude variables between male and female broilers. Males grow faster and usually experience more problems with ascites, sudden death syndrome and leg problems.

The experimental unit at Earlybird Farm (Pty) Ltd, Standerton was used because it was readily available. It is at 1517 m above sea level, which made it ideal for research on ascites on the Highveld of

South Africa. At this altitude and together with the fact that the experiment was carried out during winter (June and July), no inducing methods were necessary. The conditions in the experimental house mimicked farm conditions.

3.2. EXPERIMENTAL DESIGN

Nineteen thousand and eight eggs were placed together in a setter and transferred to a hatcher. The chickens were feather sexed and 6000 males were selected by systematic random sampling for the experiment. Two thousand chickens per treatment group were selected by systematic random sampling.

The experiment was an 8 x 3 block design. Eight replications of each treatment with 250 broilers were randomly and equally assigned to the pens in the experimental unit. Male broilers were housed under identical environmental conditions and all pens received the same starter, finisher and post finisher diets, but of three different textures, as follows:

Treatment group 1: Crumbled starter, pelleted finisher, pelleted post finisher and pelleted post finisher non medicated.

Treatment group 2: The same rations as above, but crumbles and pellets were grounded.

Treatment group 3: All mash starter, mash finisher, mash post finisher and mash post finisher non medicated.

The feeding procedure was as follows: 800g starter (∀ day 0 to day 21), 800g finisher (∀ day 22 to day 28), 1600g post finisher (∀ day 29 to day 35) and approximately 800g non medicated post finisher feed (∀ day 36 to day 42).

3.3. EXPERIMENTAL PROCEDURES

3.3.1 Serology

Serum samples were taken from 24 birds per treatment group on day 28 and 42. Three birds per pen were bled. The serum was used to serologically rule out exposure to Newcastle disease (NCD), Infectious Bronchitis (IB), Pneumovirus, Reovirus (REO), *Mycoplasma gallisepticum* (MG) and *Mycoplasma synoviae* (MS).

For NCD the Haemagglutination – Inhibition (HI) test was used. The method was according to Hitchner, Domermuth, Purchase and Williams (1980). Four HA units were used for the HI test. Log 2 serial dilutions were made on the test sera.

The test was interpreted as follows:

The serum of broilers with immunity usually test from 2^4 and higher. Lower than 2^4 is usually not protective against NCD. With a NCD challenge boilers usually test $>2^6$ (Buys, 1997).

The commercial IDEXX enzyme immunoassay (ELISA) test kit was used for detection of serum antibodies to IB, Pneumovirus or Turkey

rhinotracheitis and REO. The method used was as described by IDEXX. The optical density (OD) readings were converted to titers by means of regression lines, with software supplied by IDEXX.

According to IDEXX, titers greater than 3000 should be considered positive and indicate vaccination or exposure to these viruses. At Earlybird Farm (Pty) Ltd, IDEXX ELISA tests were usually interpreted as follows (Buys, 1997):

TRT	:	>1000	=	exposure to the virus
IB	:	>3000	=	exposure to the virus
REO	:	>6000	=	exposure to the virus

The Rapid Plate Agglutination Test was used for MG and MS. The method and interpretation were according to Hitchner *et al*. (1980).

3.3.2 Mortality

Mortalities were recorded daily.

Dead chickens were weighed individually and the weight for each chicken was recorded.

Post mortems on dead chickens were done daily. The cause of death was recorded individually. The following causes of death were recorded:

3.3.2.1 Yolk sac infection (Mushy chick disease, Omphalitis)

Yolk sac infection is characterized by bacterial infection of the naval and / or yolk sac in chickens.

3.3.2.2 Non starter (Starve out)

Non starter refers to birds with no observable infectious disease. They are usually smaller than the average chicken and die of dehydration and nephrosis.

3.3.2.3 Vaccine reaction

Vaccine reaction refers to an adverse reaction after spraying birds with a live virus vaccine (e.g. IB and NCD). It is usually characterized by a tracheitis and plugs in the trachea and / or bronchi.

3.3.2.4 Septicaemia

Septicaemic lesions included one or more of the following: air sacculitis, peritonitis, perihepatitis and pericarditis.

3.3.2.5 Sudden death syndrome (Flip-over disease, Heart attack, Lung oedema, Dead in good condition)

SDS was defined as sudden mortality in apparently healthy, fast growing broilers that die suddenly exhibiting a short wing-beating convulsive attack. SDS was described in Chapter II.

3.3.2.6 Ascites

For this experiment the term ascites specifically referred to ascites caused by pulmonary hypertention and right ventricular failure as described in Chapter II.

3.3.2.7 Skeletal disorders

Skeletal disorders were divided into infectious and non-infectious leg problems.

- **Infectious leg problems**

Birds showing infection of the hock joint and/or femur head necrosis were said to have infectious leg problems.

- **Non-infectious leg problems**

Birds showing VVD and TD were classified as non-infectious leg problems.

3.3.2.8 Other

Other disorders included aspergillosis, spondylolisthesis, injury, intestinal torsion and liver rupture.

3.3.3 Bacteriology

Specimens for bacteriological examination were collected from chickens that died of yolk sac infection, septicaemia and infection of the hock joint. Sterile cotton swabs were used for chickens with yolk

sac infection and septicaemia. In birds with infection of the hock joint the skin was burnt, an incision with a sterile scalpel was made and a sterile platinum loop was used to collect puss.

The samples were transferred onto blood agar and McConkey agar and incubated for 24 hours at 37°C. The blood agar was placed in a microaerobic atmosphere (candle jar). *Pasteurella*, *Staphylococcus*, *E.coli*, *Ornithobacterium rhinotracheale* and *Haemophilus* grow better under these conditions.

The isolates were submitted to the Onderstepoort Veterinary Institute's bacteriology section for verification.

3.4. OBSERVATIONS AND ANALYTICAL PROCEDURES

3.4.1 Bodyweight

The bodyweight of all the chickens were determined (batch weight) at day old, day 7, 14, 21, 28, 35 and 42. The average weight per treatment group was determined.

On day 4, 11, 18, 25, 32 and 39 a minimum of 50 chickens (20%) per pen were weighed (batch weight) in order to calculate the average weight per treatment group for these days. These chickens were selected by systematic random sampling.

3.4.2 Feed conversion (FC)

The amount of feed consumed by the chickens was determined on day 7, 14, 21, 28, 35 and 42. Together with the weight of the chickens on these days it was used to calculate the feed conversion per treatment group (FC is the unit of feed consumed per unit of live mass). A mortality corrected FC was also determined. The weight of the dead birds in every treatment group for every week was calculated and added to the live weight on day 7, 14, 21, 28, 35 and 42. The total feed consumed was divided by the total weight on the days mentioned.

3.4.3 Production efficiency factor (PEF)

The PEF was calculated by using the following formula:

$$PEF = \frac{\text{Mean live mass (kg)} \times \% \text{ Survivors} \times 100}{\text{Feed conversion} \times \text{Age (days)}}$$

3.5 EXPERIMENTAL ANIMALS

Table 3.5.1 describes the characteristics of the chickens used for this experimental study. Six thousand day old Ross 788 male broiler chickens were selected by systematic random sampling, from a specific broiler breeder flock (37 weeks old). The chickens were all hatched from the same setter and hatcher. The Broiler breeder flock was serologically Mycoplasma-free on rapid plate agglutination.

Table 3.5 Description of experimental animals

Animal	Poultry	
Type	Broiler chicken	
Breed/Strain	Ross 788	
Sex	Male	
Description	Broiler chickens from Mycoplasma-free breeder flock	
Origin	Earlybird Farm (Pty) Ltd Standerton	
Initial Age	Day old chickens (day 0)	
Weight	Treatment group 1	42,8 gram
	Treatment group 2	43,3 gram
	Treatment group 3	43,1 gram
Parent Flock Age	37 weeks	
Placement date	06/06/1997	
Slaughtering date	18/07/1997	

3.5.1 CONDITIONS OF HOUSING AND MANAGEMENT

3.5.1.1 Number of pens in the house

24

3.5.1.2 Size of pens

3m x 4m = 12 m²

3.5.1.3 Birds per pen

250

3.5.1.4 Stocking density

20,83 birds per m².

3.5.1.5 Number of feeders per pen

Six manual hanging tube feeders and 4 scratch trays until 10 days.

3.5.1.6 Number of drinkers per pen

Three manual hanging bell type drinkers and 6 chick fonts until 5 days.

3.5.1.7 Availability of feed and water

Ad libitum

3.5.1.8 Bedding material

Clean pine wood shavings.

3.5.1.9 Lighting programme

23 hours light and 1 hour dark.

3.5.1.10 Light intensity

Day old - 7 days : 23,7 – 26,0 lux.

8 days - 42 days : 4,3 – 7,0 lux.

The light intensity in the house was monitored weekly using a lux metre. The light intensity was measured in six of the twenty-four pens. These six pens comprise of two pens in the front, two pens in the middle and two pens at the back of the house. The measurements were taken at the level of the bedding at three different areas (front, middle and back) in the pen. The three readings were used to work out the average light intensity for each pen.

3.5.1.11 Ventilation system

Cross flow fan assisted ventilation.

3.5.1.12 Measurements of oxygen (O_2), carbon dioxide (CO_2) and ammonia (NH_3)

A Dräger multi gas detector Model 31, distributed by Dräger SA (Pty) Ltd, with the appropriate tubes were used to measure O_2, CO_2, and NH_3 on day 8, 12, 17, 21, 26, 31 and 36. For O_2 n = 1 was used, for CO_2 n = 5 and for NH_3 n = 10. The letter "n" indicates the amount of times the Dräger Multi-gas detector was pumped to measure O_2, CO_2, and NH_3. The concentrations of O_2, CO_2, and NH_3 were measured weekly, between 8:00 and 10:00 in the morning, in the same pen, in the middle of the house, at the level of the bedding.

3.5.1.13 Location of experimental facility

The six thousand broilers were housed in Earlybird Farm's experimental environmental controlled house, located at Standerton, on the Mpumalanga Highveld. The facilities are located at an altitude of 1517 metres above sea level.

3.5.1.14 Basal ration

Starter, finisher and post finisher ration were representative of local formulations. An anticoccidial drug (Elancoban from Elanco) and growth promotant (Pit-Bac from Feed Ad) were included in the ration to mimic standard commercial practice.

A steam-pressure-die process was used to pellet the feeds. It was done at 85°C. The pellet diameter was 4,5

mm and the length of the pellet was approximately 10,0 mm. To make the crumbles the pellets were put through a crumbler, with the roller gap at 1,7 mm. For the ground crumbles and pellets the roller gap was 1,0 mm. For the mash, a commercial-type hammer mill, with a screen size of 8 mm, was used.

3.5.1.15 Feeding Programme

800g Starter
800g Finisher
1600g Post finisher
± 800g Post finisher non medicated
The rations were fed *ad libitum*.

3.5.1.16 Size of feed particles

Treatment group I received starter crumbles, pelleted finisher, pelleted post finisher and pelleted post finisher non medicated feed. For Treatment group 2, crumbles and pellets were grounded. Treatment group 3 received all-mash starter, mash finisher, mash post finisher and mash post finisher non medicated feed.

The size of the three different types of feed was measured by the Animal Research Council, Irene. The size of the feed particles was measured with laboratory test sieves (BS 410/1986) from Endocotts LTD, London, England.

3.5.1.17 Water quality

Water was potable and within biological norms for animal consumption.

3.5.1.18 Hygiene management

The house was washed, disinfected and fumigated before placement of the chickens.

3.5.1.19 Biosecurity

The facilities were fenced off and visitors and workers showered and wore protective clothing.

3.5.1.20 Vaccination history

Day old (hatchery)	NCD oil subcutaneously, cloned La Sota and IB H120 (course spray).
Day 7	TRT (course spray).
Day 12	Intermediate Infectious Bursal Disease (IBD) strain (drinking water).
Day 18	Cloned La Sota and IB H120 (course spray).

3.5.1.21 Transportation

The day old chickens were transported from the hatchery to the experimental house with one of Earlybird Farm's hatchery trucks. The journey took about 10 minutes. The 42 day-old broilers were transported to the abattoir with Earlybird Farm's trucks. The journey was approximately 15 minutes.

3.5.1.22 Environmental temperature

The broiler house was heated with one gas heater fitted with a plastic windsock. For the first ten days fans and ventilation openings were closed with plastic to keep the

house warm. The experiment was done during the winter from 06/06/1997 to 18/07/1997.

3.5.1.23 Recording of temperature and relative humidity

A min/max thermometer and hygrothermograph were placed in the poultry house, as well as outside. Outside, the thermometer and hygrothermograph were placed in a Stevenson screen.

CHAPTER IV

4. RESULTS AND DISCUSSION

4.1 SEROLOGY

For NCD a Haemagglutination-Inhibition test was used. For IB, Pneumovirus and REO an IDEXX ELISA test kit was used and for MG and MS a Rapid Plate Agglutination Test was done. The results are summarized in Table 4.1.1 to Table 4.1.5.

Table 4.1.1 Newcastle Disease Haemagglutination-Inhibition results on day 28 and day 42

Treatment Group	28 d	Coefficient of variation (CV %)	42 d	Coefficient of variation (CV %)
1	2,4	54,8	3,7	19,5
2	2,8	52,9	4,1	25,8
3	3,3	39,4	3,6	24,8

Table 4.1.2 Infectious Bronchitis IDEXX ELISA results on day 28 and day 42

Treatment Group	28 d	Coefficient of variation (CV %)	42 d	Coefficient of variation (CV %)
1	301	29,8	502	27,6
2	389	36,1	603	37,7
3	302	35,4	575	32,1

Table 4.1.3 Turkey Rhinotracheitis IDEXX ELISA results on day 28 and day 42

Treatment Group	28 d	Coefficient of variation (CV %)	42 d	Coefficient of variation (CV %)
1	219	62	174	126
2	194	88	245	111
3	162	64	219	145

Table 4.1.4 Reovirus IDEXX ELISA results on day 28 and day 42

Treatment Group	28 d	Coefficient of variation (CV %)	42 d	Coefficient of variation (CV %)
1	768	166	5590	104
2	610	175	1601	91
3	875	140	3862	97

Table 4.1.5 MG and MS Rapid Plate Agglutination Test results on day 28 and day 42

Treatment Group	Test	28 d	42 d
1	MG	Negative	Negative
1	MS	Negative	Negative
2	MG	Negative	Negative
2	MS	Negative	Negative
3	MG	Negative	Negative
3	MS	Negative	Negative

Discussion

The HI titers against Newcastle Disease varied between 2.4 and 3.3 at 28 days and between 3.6 and 4.1 at 42 days. These titers are normal after vaccination with a lentogenic Newcastle vaccine (Buys, 1997).

The IDEXX ELISA titers against Infectious Bronchitis varied from 301 to 389 at 28 days and from 502 to 603 at 42 days. According to IDEXX this titers exclude a challenge by an IB virus.

The Turkey Rhinotracheitis ELISA titers are normal and were between 162 and 219 at 28 days and between 174 and 245 at 42 days. There is no indication of a challenge with a Pneumovirus (IDEXX).

The Reovirus ELISA results were between 610 and 875 at 28 days and between 1601 and 5590 at 42 days. Although the titers for treatment group 1 and 3 are higher than that of treatment group 2, no clinical disease was observed in any of the groups. At Earlybird Farm (Pty) Ltd titers above 6000 indicated a possible challenge with Reovirus (Buys, 1997).

The Rapid Plate Agglutination Test against *M. gallisepticum* and *M. synoviae* was negative.

The results of the serology confirms that there were no interference by NCD, IB, Pneumovirus, Reovirus, MG or MS.

4.2 BODYWEIGHT

The average bodyweight for each treatment group on the days the chickens were weighed, is summarized in Table 4.2.1. Figure 4.2.1 illustrates the data from Table 4.2.1 graphically and also shows the growth profile over 42 days for the different treatment groups.

The weekly weight gain for each treatment group is given in Table 4.2.2 and graphically illustrated in Figure 4.2.2.

Discussion

Chickens on the crumble-pellet dietary regimen (2304.0 g on day 42) were significantly (p#0,05) heavier at 42 days when compared with birds fed either all-mash (2054.1g on day 42), or fed the ground crumble-pellet regimen (1993.5g on day 42). The difference in bodyweight between chickens fed all-mash and ground crumbles and pellets is significant (p#0,05) (Appendix I – Table 5.1). The heavier bodyweight exhibited by birds fed the crumble-pellet regimen compared with those on the all-mash and the ground crumble-pellet regimen is consistent with earlier reports (Proudfoot *et al*., 1982b and Plavnik *et al*., 1997).

The difference in bodyweight between the treatment groups was probably caused by the size of the feed particles. See Table 4.9.1. In the ground crumble-pellet diet 41,5% of the feed particles were smaller than 0,6 mm, in the all-mash diet 25,0%, in the crumbled diet 11,0%, and in the pelleted diet only 5,0% of the feed particles were smaller than 0,6mm. In the ground crumbles and pellets only 3,5% of the particles were greater than 3,6 mm and in the mash diet 7,5%, compared to 44% of the crumbles and 76,7% of the pellets.

This indicates a good correlation between feed particle size and bodyweight during broiler grow-out, as well as final bodyweight at 42 days of age.

The three treatment groups received feed with the same specification and two groups received feed that went through the pelleting process. The chickens that received the ground crumbles and pellets, with the most small particles, recorded the lowest bodyweight from 7 to 42 days of age. Bodyweight is therefore determined by feed particle size and not by the pelleting process.

The weekly weight gain in grams per week, in all three treatment groups, increased weekly until 35 days of age. During week 6 the weight gain in grams per week was lower if compared to week 5 in all three treatment groups. This was probably due to increased stress, caused by less space for each chicken and a decrease in feeding and drinking space.

The percentage weight gain per week, decreased from week one to week six. During week one and to a lesser extent during week two there was a big difference in percentage weight gain between treatment groups. The chickens on the crumbles and pellets grew the fastest (230,1% during week one and 159% during week two). The percentage weight gain for the chickens on the all-mash diet was 187,7% for week one and 153,1% for week two. The slowest percentage weight gain was in the chickens on the ground crumbles and pellets (179,7% during week one and 143,5% during week two).

The difference in percentage weight gain over the first two weeks, was probably due to the difference in feed texture, because the chickens on the crumbles grew the fastest and the chickens on the ground crumbles the slowest. The improvement in percentage

weight gain in treatment groups 2 (ground crumbles and pellets) and 3 (mash) when compared to treatment group 1 (crumbles and pellets), from three weeks onwards, was probably because the chickens adapted to the fine texture of the ground crumbles and pellets, and the mash. Another factor that could have influenced the percentage weight gain from week three onwards, is compensatory growth. When a broiler experiences a period of under nutrition it is usually followed by compensatory growth (Zubair and Leeson, 1996).

The percentage weight gain per week in the three treatment groups from week three to week six was similar.

Table 4.2.1 Average bodyweight in gram per treatment group from day 0 to day 42

Treat-ment group	Age (days)	Body-weight (g)	Treat-ment group	Age (days)	Body-weight (g)	Treat-ment group	Age (days)	Body-weight (g)
1	0	42,8	2	0	43,3	3	0	43,1
	4	87,2		4	81,4		4	78,1
	7	141,3		7	121,1		7	124,0
	11	250,5		11	207,4		11	214,6
	14	366,0		14	294,9		14	313,9
	18	575,5		18	461,3		18	494,5
	21	739,8		21	598,0		21	643,8
	25	1003,5		25	838,8		25	913,5
	28	1216,6		28	1007,0		28	1061,8
	32	1545,8		32	1304,0		32	1359,3
	35	1791,4		35	1506,0		35	1580,8
	39	2149,3		39	1875,5		39	1933,3
	42	2304,0		42	1993,5		42	2054,1

Figure 4.2.1 Average bodyweight in gram per treatment group from day 0 to day 42

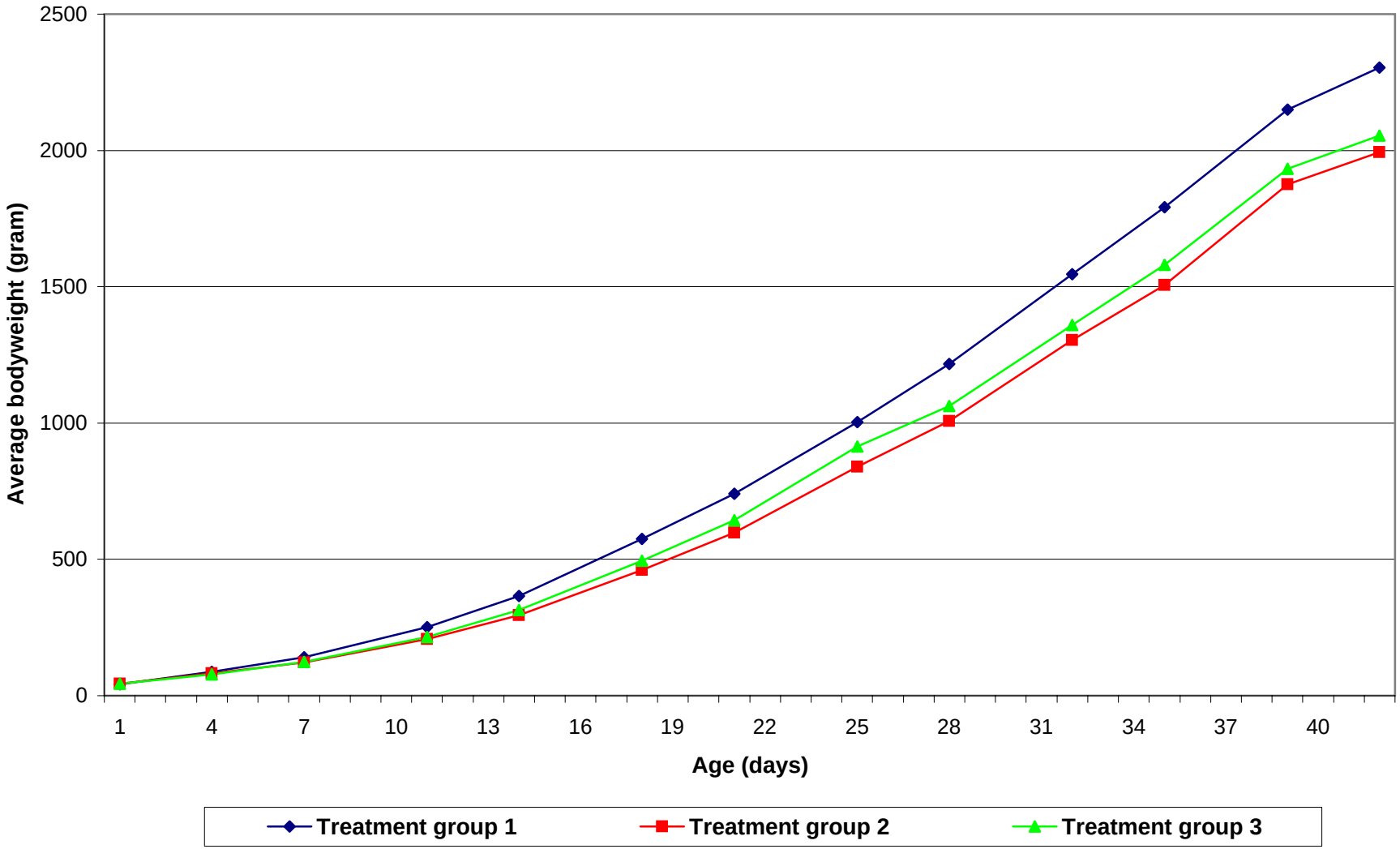


Table 4.2.2 Weekly weight gain expressed in gram and percentage per treatment group from day 0 to day 42

Treat-ment group	Age (days)	Weight gain		Treat-ment group	Age (days)	Weight gain		Treat-ment group	Age (days)	Weight gain	
		(g)	(%)			(g)	(%)			(g)	(%)
1	0 - 7	98,5	230,1	2	0 - 7	77,8	179,7	3	0 – 7	80,9	187,7
	8 - 14	224,7	159,0		8 - 14	173,8	143,5		8-14	189,9	153,1
	15 - 21	373,8	102,1		15 - 21	303,1	102,8		15 – 21	329,9	105,1
	22 - 28	476,8	64,4		22 - 28	409,0	68,4		22 – 28	418,0	64,9
	29 - 35	574,8	47,2		29-35	499,0	49,6		29 – 35	519,0	48,9
	36 - 42	512,6	28,6		36 - 42	487,5	32,4		36 – 42	473,3	29,9

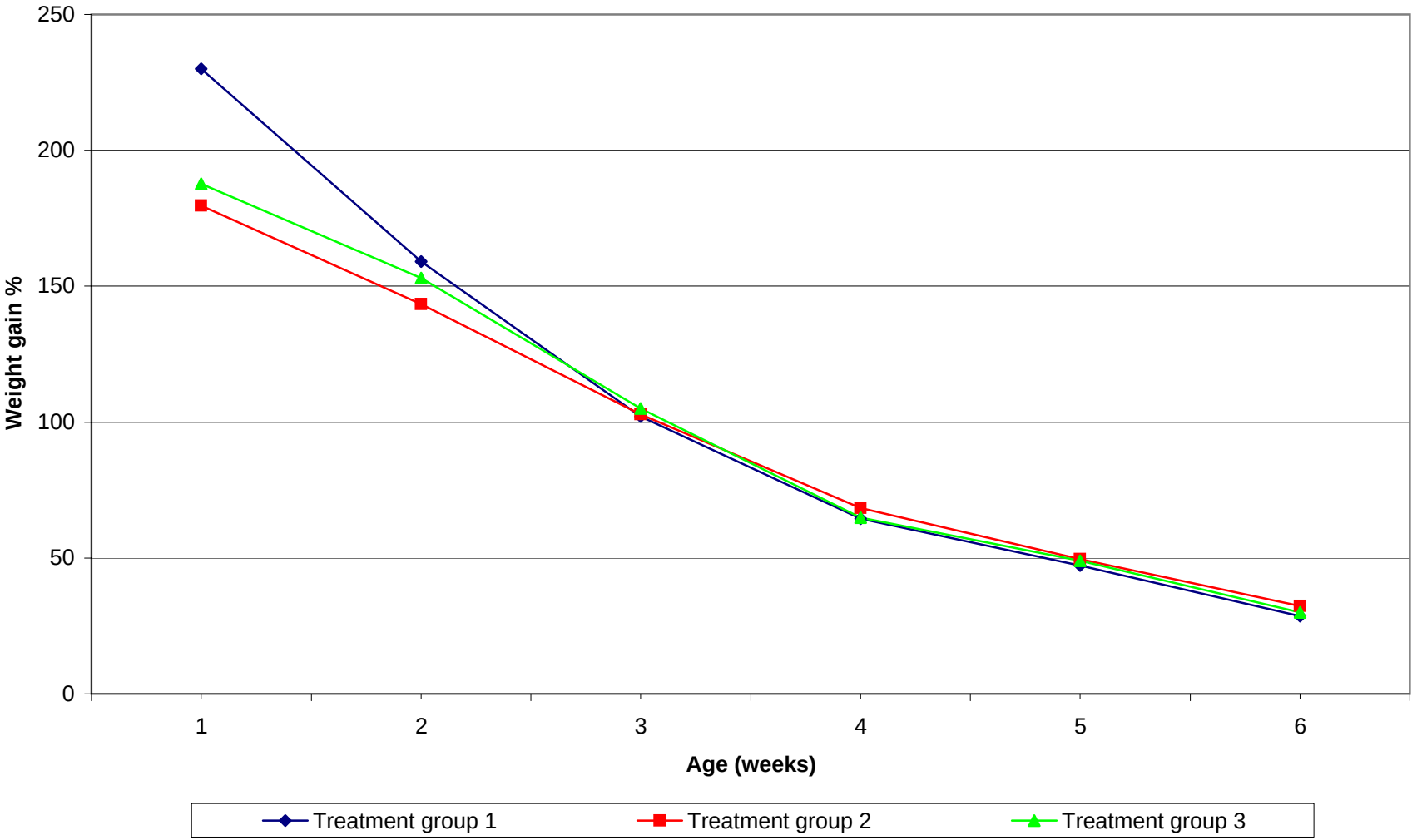
4.3. FEED CONVERSION

The feed conversion and mortality corrected feed conversion was calculated on day 7, 14, 21, 28, 35 and 42. The results are tabulated in Table 4.3.1 and graphically illustrated in Figure 4.3.1.

Discussion

Feed conversion in the chickens on the crumbles and pellets (1,900 at 42 days) was significantly (p #0,05) better than the chickens fed the ground crumble-pellet diet (1,946 at 42 days) and the all-mash diet (1,963 at 42 days). The feed conversion in treatment group 2 and 3 was not significantly (p >0,05) different (Appendix I – Table 5.2). These results agree with the finding of Proudfoot *et al*. (1982b) and Plavnik *et al*. (1997).

Figure 4.2.2 Weekly weight gain in percentage per treatment group from day 0 to day 42



The mortality corrected feed conversion in the chickens on crumbles and pellets (1,852 at 42 days) was significantly (p #0.05) better when compared to treatment group 2 and 3. The mortality corrected feed conversion in the chickens on the ground crumble-pellet diet (1,921 at 42 days) was not significantly (p>0,05) different from the chickens on the all-mash (1,945 at 42 days) diet (Appendix I – Table 5.3).

It is important to note that the mortality can make a big difference in the FC. In treatment group 1 the difference was 4,8 points with a mortality rate of 6.57 %. In treatment group 2 the difference was 2,5 points with a mortality rate of 4,03 % and in treatment group 3, 1,8 points with a mortality rate of 2,85 %.

The mortality corrected FC in the chickens on crumbles and pellets was 6,9 points better if compared to the chickens on ground crumbles and pellets and 9,3 points if compared to the chickens on mash. The difference of 2,4 points between chickens on ground crumbles and pellets, and mash was not significant (p>0,05). It means that the pelleting process on its own does not cause a significant improvement in feed efficiency, but it may play a role in the better FC on feed (crumbles and pellets, as well as ground crumbles and pellets) that went through the pelleting process.

The improvement in feed efficiency when feeding crumbles and pellets can be explained by; (a) growth promotion alone, (b) the decrease in diet volume and the large particle size, which facilitates feed or energy consumption and (c) reduced energy expenditure in the process of feed consumption.

The improvement in FC on feed that went through the pelleting process may be explained by the following; (a) inactivation of heat-

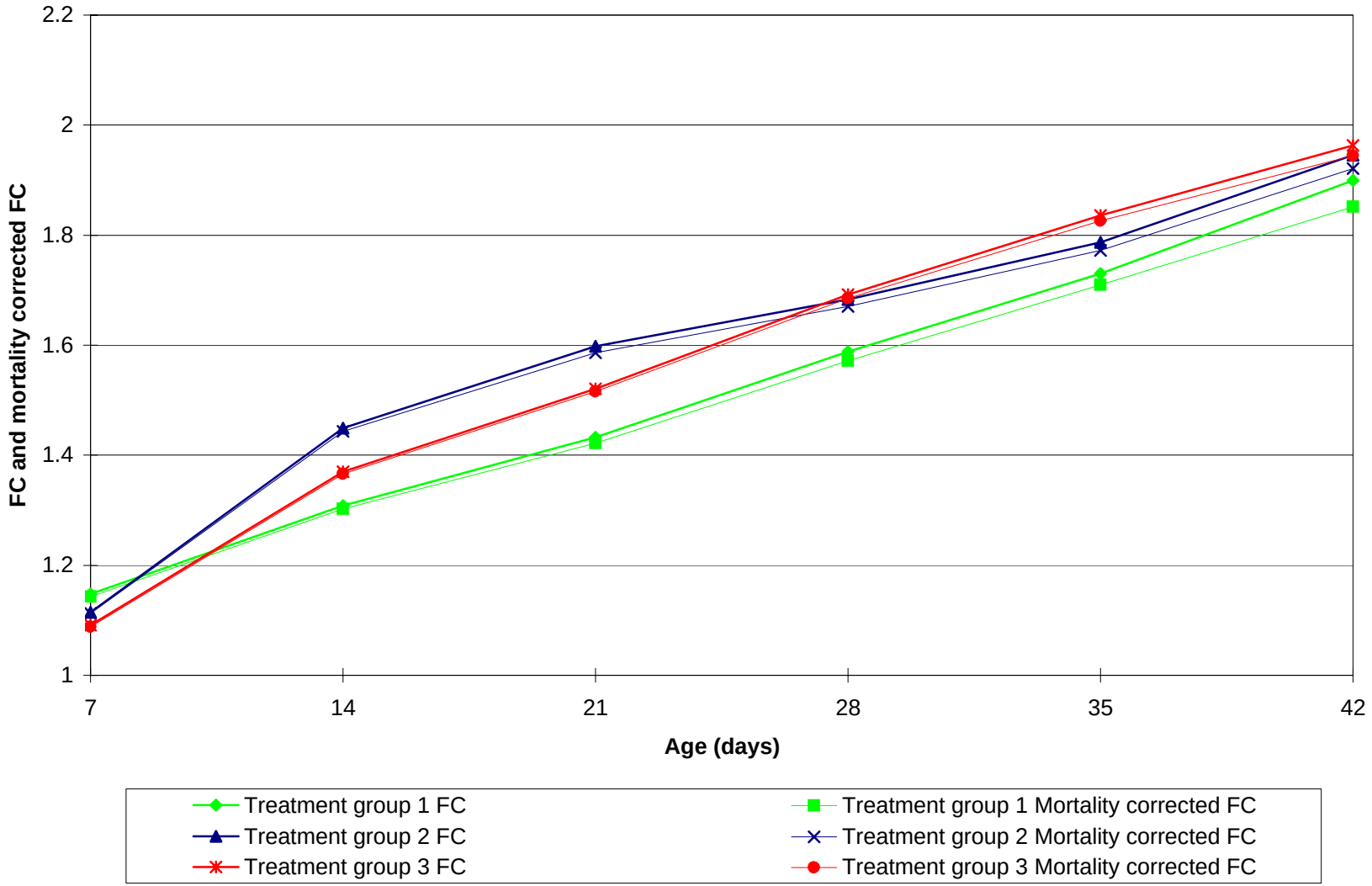
labile toxic factors in feeds, (b) improvement in digestion or utilization of feed ingredients, especially those high in fiber, (c) chemical changes in dietary carbohydrates resulting in increased metabolizable energy values, although the improvement in metabolizable energy by pelleting has not been substantiated and (d) increases in protein/amino acid bio-availability in broilers.

It can be concluded that feed texture (feed particle size) plays the most important role in determining feed efficiency in broilers.

Table 4.3.1. Weekly FC and mortality corrected FC per treatment group from day 0 to day 42

Treatment group	Age (days)	FC	Mortality Corrected FC
1	7	1,148	1,143
	14	1,308	1,302
	21	1,433	1,422
	28	1,588	1,572
	35	1,730	1,710
	42	1,900	1,852
2	7	1,115	1,113
	14	1,449	1,443
	21	1,598	1,586
	28	1,683	1,670
	35	1,787	1,772
	42	1,946	1,921
3	7	1,091	1,088
	14	1,370	1,366
	21	1,521	1,516
	28	1,692	1,685
	35	1,836	1,826
	42	1,963	1,945

Figure 4.3.1 Weekly FC and mortality corrected FC per treatment group from day 0 to day 42



4.4 MORTALITY

Mortalities were recorded and necropsied daily. The cumulative mortality and average weekly mortality for treatment groups 1, 2 and 3 are tabulated in Table 4.4.1. and Table 4.4.2. and graphically illustrated in Figure 4.4.1. and Figure 4.4.2.

The causes of the mortalities are summarized in Table 4.4.3 and in Figure 4.4.3. The most important causes of the mortalities were SDS, ascites, leg problems and septicaemia. The weekly incidence of the above mentioned causes is tabulated in Table 4.4.4 to Table 4.4.7 and graphically illustrated in Figure 4.4.4 to Figure 4.4.7. In Table 4.4.8 the causes of infectious and non-infectious leg problems and the legs affected are illustrated.

The bacteria isolated from samples that were taken from the chickens that died of septicaemia were in 82 % of samples *E. coli*, 5 % of samples *Proteus*, 5 % of samples *Staphylococcus epidermidis*, 5% of samples *Enterococcus* and 3 % of samples *Pseudomonas*. *E. coli* was isolated out of the hock joints of all the chickens that died due to infectious leg problems. It was not necessary to take any samples for virology and histopathology.

Table 4.4.1 Cumulative weekly mortality percentage per treatment group from day 0 to day 42

Treat-ment group	Age (days)	Mortality (%)	Treat-ment Group	Age (day)	Mortality (%)	Treat-ment group	Age (day)	Mortality (%)
1	7	0,82	2	7	0,39	3	7	0,60
	14	1,54		14	1,03		14	0,76
	21	2,52		21	2,10		21	1,21
	28	3,34		28	2,50		28	1,47
	35	4,26		35	3,00		35	2,04
	42	6,57		42	4,03		42	2,85

Figure 4.4.1 Cumulative weekly mortality percentage per treatment group from day 0 to day 42

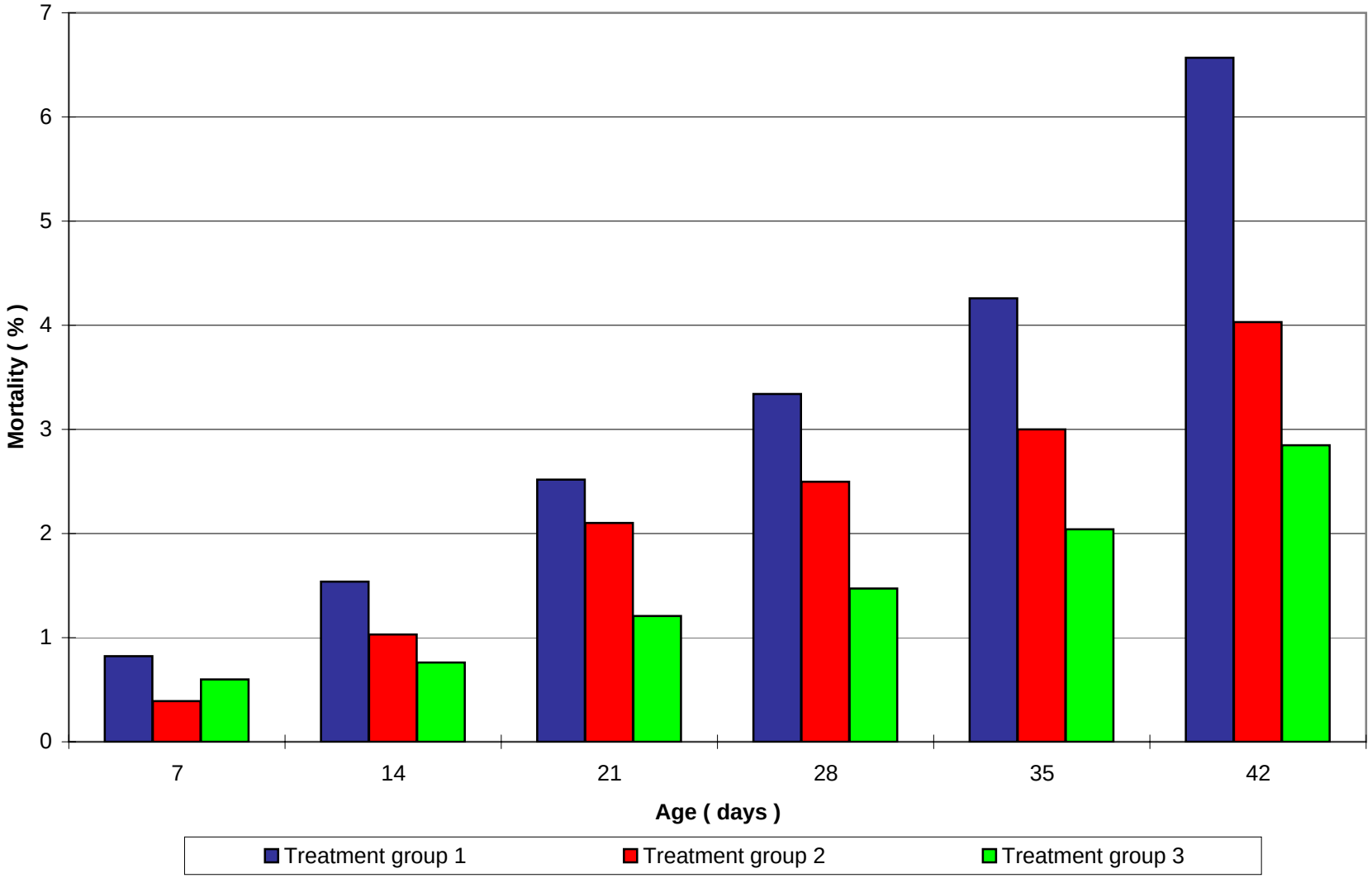


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Table 4.4.2 Average weekly mortality percentage per treatment group from day 0 to day 42

Treat-ment group	Age (days)	Mortality (%)	Treat-ment group	Age (days)	Mortality (%)	Treat-ment group	Age (days)	Mortality (%)
1	0 - 7	0,82	2	0 - 7	0,39	3	0 - 7	0,60
	8 - 14	0,72		8 - 14	0,64		8 - 14	0,15
	15 - 21	0,98		15 - 21	1,05		15 - 21	0,46
	22 - 28	0,82		22 - 28	0,41		22 - 28	0,25
	29 - 35	0,92		29 - 35	0,51		29 - 35	0,56
	36 - 42	2,31		36 - 42	1,03		36 - 42	0,83

Table 4.4.3 Causes of mortality in treatment group 1, 2 and 3 from 0 to 42 days of age

Causes of mortality	Mortality (%)			% of total mortality		
Treatment Group	1	2	3	1	2	3
Yolk sac infection	0,41	0,20	0,46	6,23	5,00	16,14
Vaccine reaction	0,36	0,15	0,10	5,47	3,72	3,51
Starve out	0,36	0,60	0,10	5,47	14,89	3,51
Sudden death syndrome	1,39	1,01	0,51	21,12	25,06	17,89
Ascites	2,11	0,10	0,41	32,07	2,48	14,39
Legs non infectious	0,41	0,81	0,25	6,23	20,10	8,77
Legs infectious	0,26	0,15	0,31	3,95	3,72	10,88
Septicaemia	0,87	0,71	0,56	13,22	17,62	19,65
Other	0,41	0,30	0,15	6,23	7,44	5,26

Figure 4.4.2 Average weekly mortality percentage per treatment group from day 0 to day 42

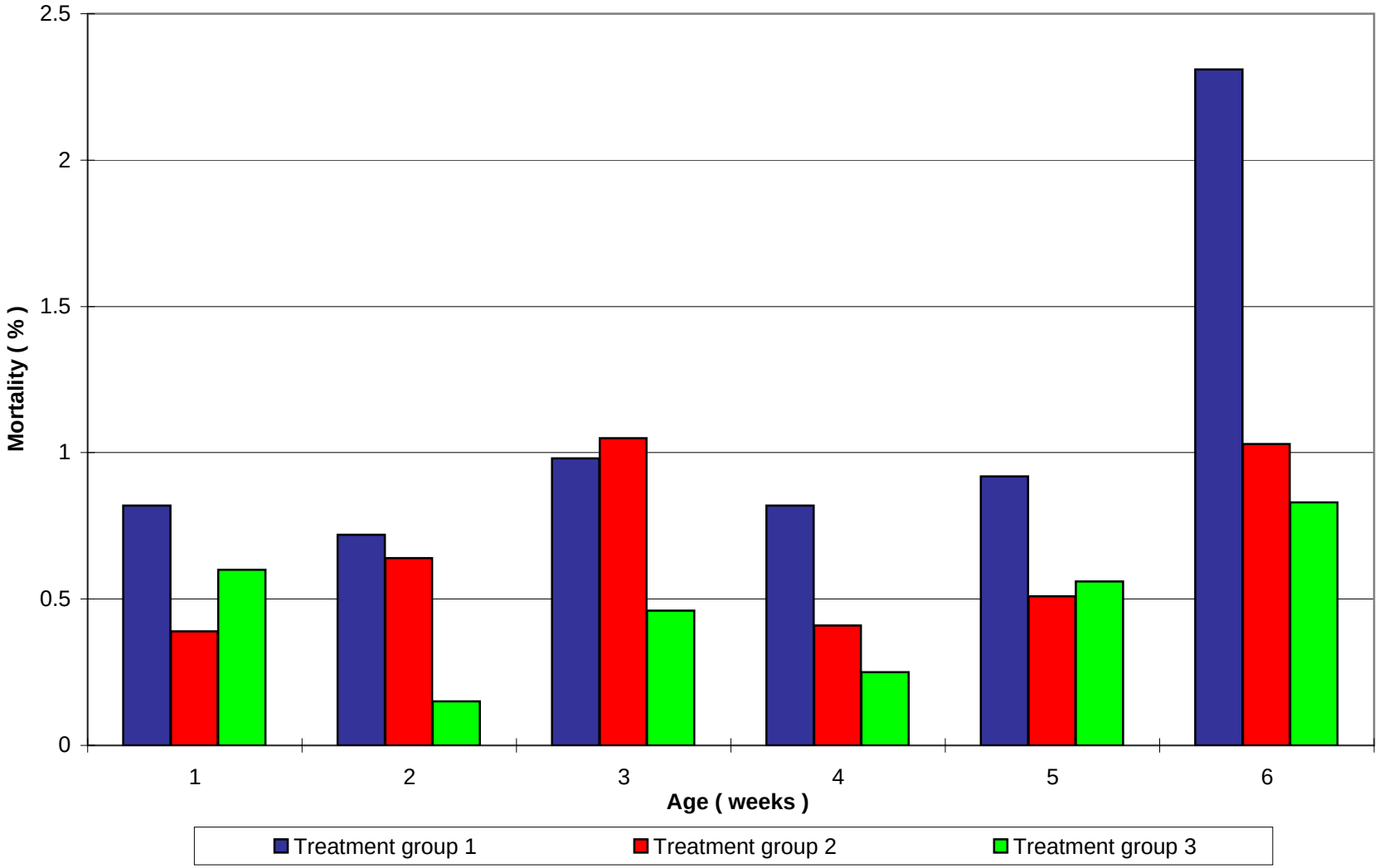

Figure 4.4.3 Causes of mortality in treatment group 1, 2 and 3 from day 0 to day 42

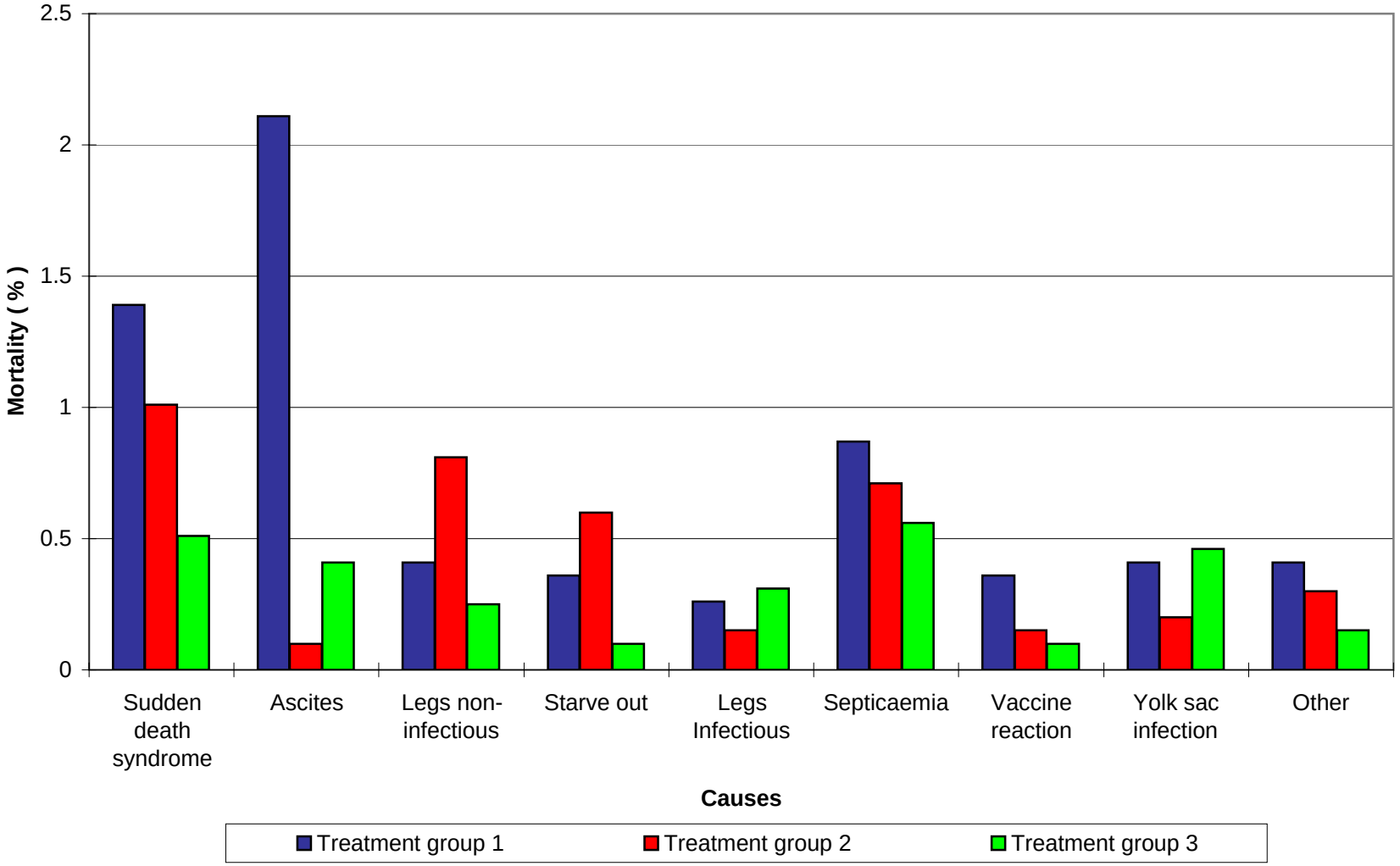

Table 4.4.4 Weekly incidence of Sudden Death Syndrome from 0 to 42 days of age

Treat-ment group	Age (days)	Mortality (%)	Treat-ment group	Age (days)	Mortality (%)	Treat-ment group	Age (days)	Mortality (%)
1	0 - 7	0,05	2	0 - 7	0,05	3	0 - 7	0,05
	8 - 14	0,15		8 - 14	0,25		8 - 14	0,05
	15 - 21	0,40		15 - 21	0,40		15 - 21	0,15
	22 - 28	0,26		22 - 28	0,26		22 - 28	0,15
	29 - 35	0,26		29 - 35	0,05		29 - 35	0,00
	36 - 42	0,26		36 - 42	0,05		36 - 42	0,10

Table 4.4.5 Weekly incidence of Ascites from 0 to 42 days of age

Treat-ment group	Age (days)	Mortality (%)	Treat-ment group	Age (days)	Mortality (%)	Treat-ment group	Age (days)	Mortality (%)
1	0 - 7	0,00	2	0 - 7	0,00	3	0 - 7	0,00
	8 - 14	0,10		8 - 14	0,00		8 - 14	0,00
	15 - 21	0,15		15 - 21	0,00		15 - 21	0,05
	22 - 28	0,15		22 - 28	0,00		22 - 28	0,05
	29 – 35	0,31		29 - 35	0,00		29 - 35	0,10
	36 – 42	1,53		36 - 42	0,10		36 - 42	0,26

Figure 4.4.4 Weekly incidence of Sudden Death Syndrome from day 0 to day 42

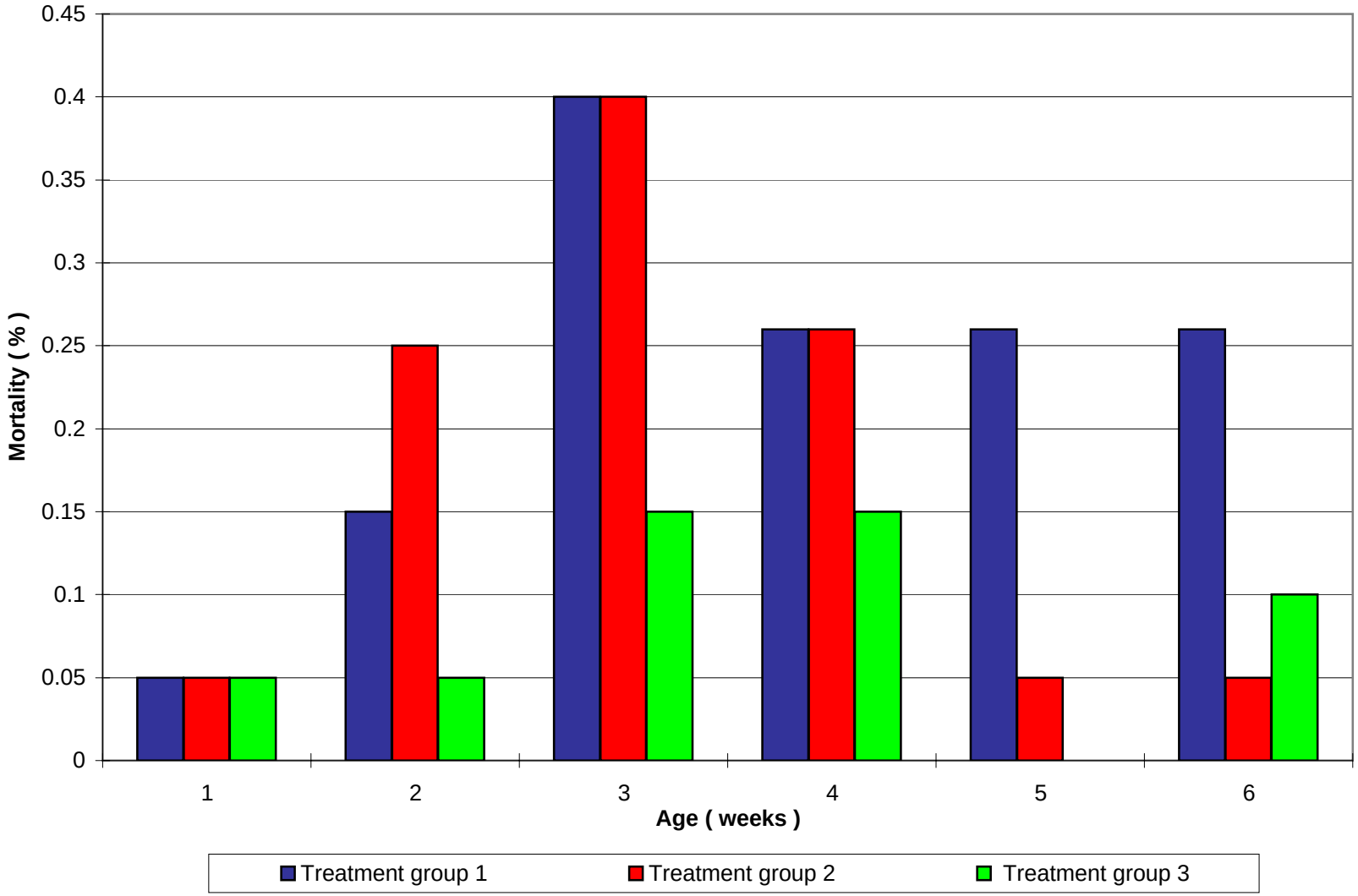

Figure 4.4.5 Weekly incidence of Ascites from day 0 to day 42

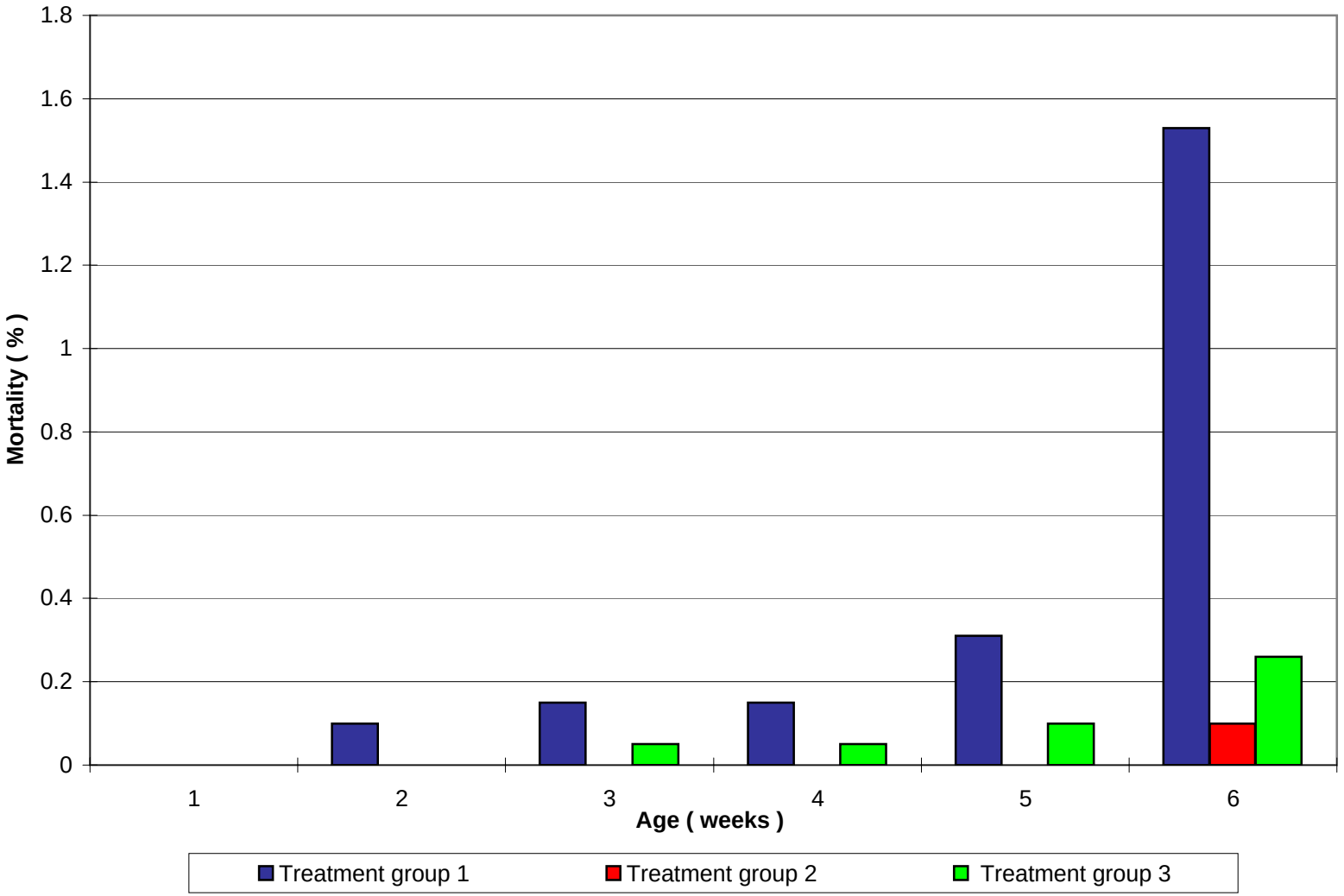

Table 4.4.6 Weekly incidence of Leg Problems (infectious and non-infectious) from 0 to 42 days of age

Treat-ment group	Age (days)	Mortality (%)	Treat-ment group	Age (days)	Mortality (%)	Treat-ment group	Age (days)	Mortality (%)
1	0 – 7	0,0	2	0 - 7	0,0	3	0 - 7	0,0
	8 –14	0,0		8 - 14	0,0		8 - 14	0,0
	15 – 21	0,05		15 - 21	0,15		15 - 21	0,15
	22 – 28	0,1		22 - 28	0,05		22 - 28	0,0
	29 – 35	0,21		29 - 35	0,26		29 - 35	0,15
	36 – 42	0,32		36 - 42	0,52		36 - 42	0,31

Table 4.4.7 Weekly incidence of Septicaemia from 0 to 42 days of age

Treat-ment group	Age (days)	Mortality (%)	Treat-ment group	Age (days)	Mortality (%)	Treat-ment group	Age (days)	Mortality (%)
1	0 – 7	0,0	2	0 - 7	0,0	3	0 - 7	0,0
	8 – 14	0,05		8 - 14	0,05		8 - 14	0,05
	15 – 21	0,1		15 - 21	0,15		15 - 21	0,0
	22 – 28	0,31		22 - 28	0,1		22 - 28	0,05
	29 – 35	0,16		29 - 35	0,1		29 - 35	0,21
	36 – 42	0,26		36 - 42	0,31		36 - 42	0,26

Figure 4.4.6 Weekly incidence of Leg Problems (infectious and non-infectious) from day 0 to day 42

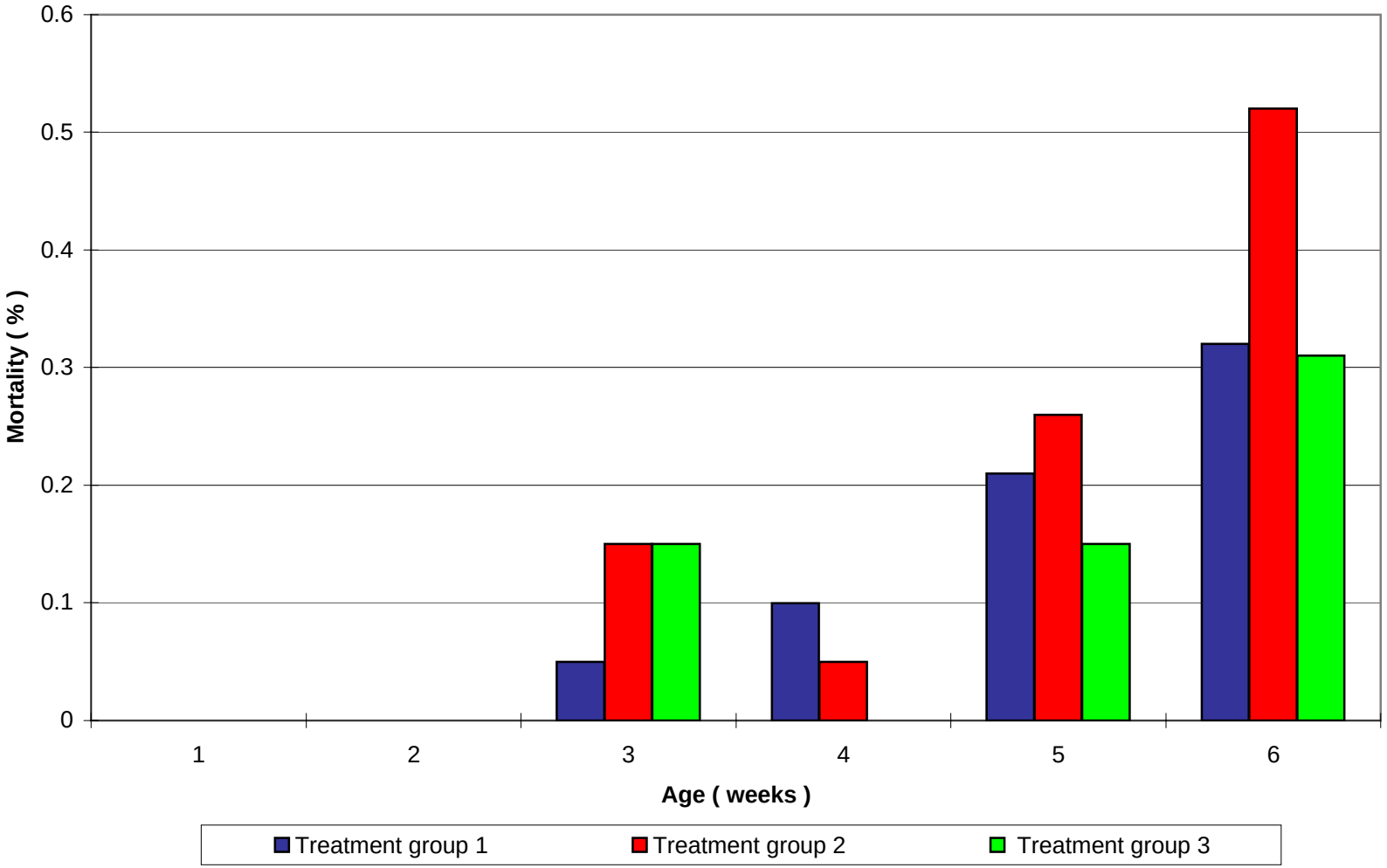

Figure 4.4.7 Weekly incidence of Septicaemia from day 0 to day 42

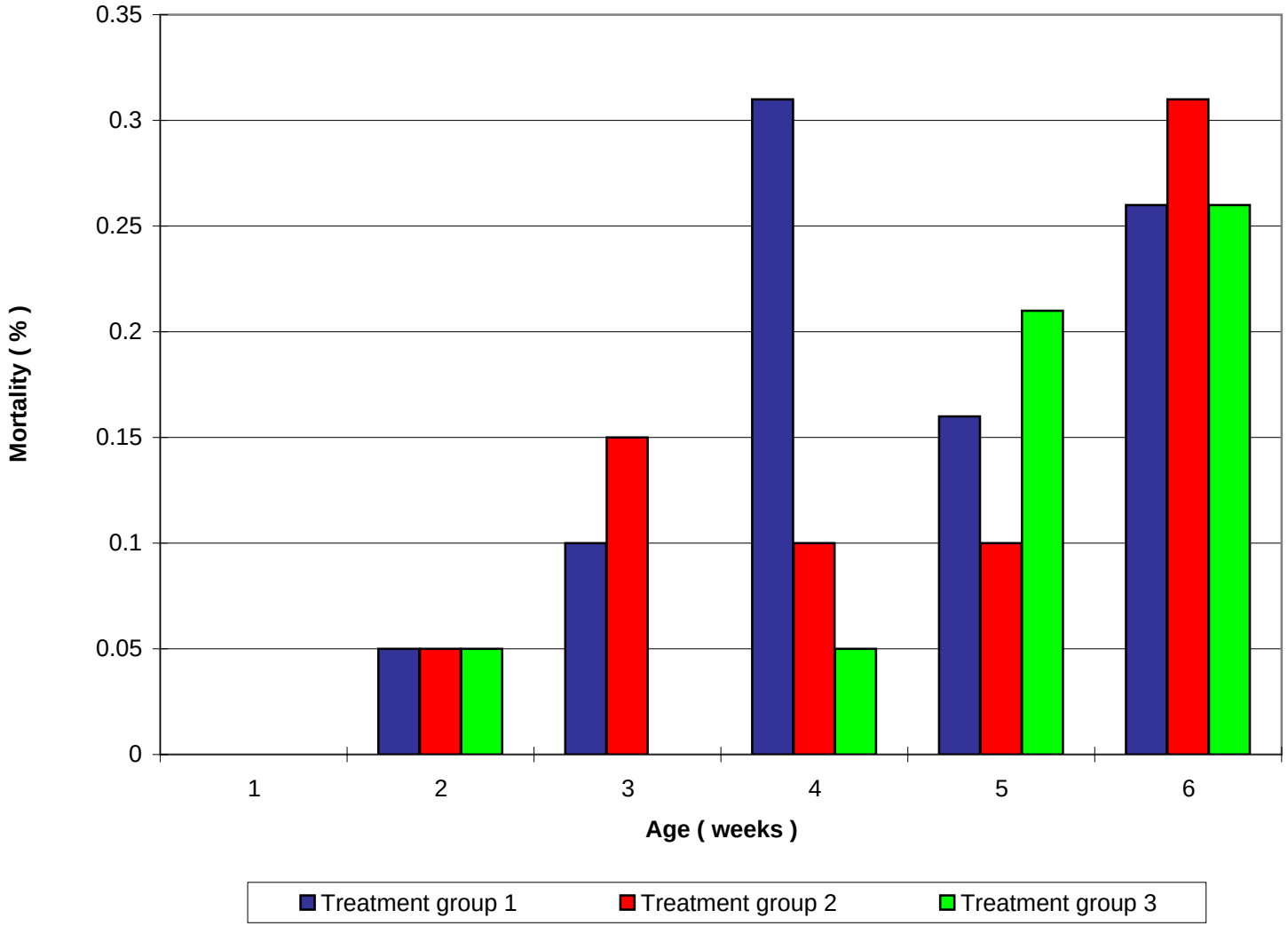

Table 4.4.8 The causes of infectious and non-infectious Leg Problems and the leg(s) affected

Treat-ment group	Infectious			Non-infectious					
				Valgus-Varus Deformation			Tibial Dyschondroplasia		
	Left leg	Right leg	Both legs	Left leg	Right leg	Both legs	Left leg	Right leg	Both legs
1	2	0	3	2	2	4	0	0	0
2	0	2	1	2	4	7	0	0	3
3	4	2	0	0	1	4	0	0	0
Total	6	4	4	4	7	15	0	0	3

Discussion

Mortality was significantly (p #0,05) higher in chickens fed the crumble-pellet regimen (6,57% at 42 days), compared to chickens on the ground crumbles and pellets (4,03% at 42 days) and all-mash regimen (2,85% at 42 days). The higher mortality was mainly caused by ascites (2,11%) and SDS (1,39%).

The mortality in chickens on the ground crumble-pellet regimen was significantly (p #0,05) higher than that of the all-mash regimen (Appendix I – Table 5.4). The higher mortality was mainly caused by SDS (1,01%) and non-infectious leg problems (0,81%).

Ascites

The incidence of ascites was significantly (p #0,05) higher in the crumble-pellet dietary regimen (2,11%) (Appendix I – Table 5.6). In the group fed ground crumbles and pellets, it was only 0,1% and in the all-mash group it was 0,41%. Broilers on crumbles and pellets were 20,5 times more likely to die from ascites than chickens on ground crumbles and pellets, and 5,13 times more likely to die from ascites than broilers on mash. The chickens on the crumbles and pellets grew the fastest and had the highest incidence of ascites. The chickens on the mash grew second fastest with the second most cases of ascites. The chickens on the ground crumbles and pellets grew the slowest with the least cases of ascites. This indicates that the pelleting process by itself does not increase mortality due to ascites. This is in accordance with Julian *et al*. (1987) who reported that rapidly growing broilers are more susceptible to increased pulmonary arterial pressure resulting in right ventricular hypertrophy, right ventricular failure and ascites than slower-growing broilers. Increased blood-flow because of rapid growth and the increased blood viscosity caused by polycythaemia because of hypoxia-induced hypoxaemia, may be the main reasons for the dramatic increase of PHS in hypoxia-induced pulmonary hypertension in broiler chickens (Julian, 1993).

Others suggested that PHS at low altitude and the increased incidence at high altitude is related to the high oxygen requirement of rapid growth and the inability of the heart and lung to deliver sufficient oxygen to the tissue to maintain genetic and nutritional growth rate potential (Julian, 1993). Julian *et al*. (1992) showed that fast-growing broilers have a lower percentage oxygen saturation than slow-growing broilers. They also reported that heavy broilers

may have hypoxic hypoxaemia because of reduced lung volume as a percentage of bodyweight, because the large breast muscle mass interferes with respiration, or because intra-abdominal pressure from fat and ingesta interferes with the bellows effect of the air sacs, reducing tidal volume.

The first case of ascites was diagnosed at 11 days. It gradually increased with the highest incidence from day 36 to 42. Of the 2,24% chickens that died due to ascites in treatment group 1 (crumbles and pellets), 1,53% died during the last week. In treatment group 3 (mash) 0,26% of the 0,46% cases of ascites occurred during the last week. In treatment group 2 (ground crumbles and pellets) mortality due to ascites occurred only during the last week (0,10%). Hassanzadeh, Ladmakhi, Buys, Dewil, Rahimi and Decuypere (1997) reported that mortality due to ascites occurred from 3 weeks of age onwards and the rate increased considerably between weeks 4 and 6.

The high percentage of ascites in the chickens on crumbles and pellets, can probably be explained by their high growth rate during the first week and to a lesser extent during the second week. The percentage weight gain for this group during the first week was 230,1% and the mortality due to ascites was 2,24%. In the chickens on mash the weight gain during the first week was 187,7% and the incidence of ascites was 0,46%. In the chickens on ground crumbles and pellets the weight gain during the first week was 179,7% and the incidence of ascites was 0,10%. This finding agrees with the research of Malan, Siebrits, Casey, Coetzer and Janse van Vuuren (1996) who reported that ascitic broiler chickens are growing significantly faster during the first week post-hatch and that slowing their growth might help to prevent, or control the incidence of

ascites. They concluded that the 5-day bodyweight of a broiler can be a useful tool in identifying ascitic individuals.

Lott, Branton and May (1996) used light restriction to restrict the growth of male broilers up to 41 days of age. Light restriction significantly reduced bodyweight at 22 days, but not at 52 days. The light-restricted birds exhibited the fastest growth from 22 days of age to the end of the trials (52 days of age). Chickens with the restricted lighting, experienced the lowest ascites mortality. Therefore, their results suggested that development of ascites may not necessarily be a function of fast growth throughout the grower diet period (22 to 52 days of age), but rather, may arise subsequent to rapid growth rate during the brooding phase (1 to 22 days of age). Had fast growth been the single determining factor, the treatment that exhibited the fastest growth from 22 – 52 days of age (light restricted at the early age) would have developed the highest incidence of mortality associated with ascites.

Lamas da Silva *et al*. (1988) conducted a study to investigate whether the use of pelleted feeds might influence the incidence of ascites in broilers reared at low altitudes (730 metres above sea level). Their data suggested that the positive relationship between pelleted feeds and incidence of ascites observed at high altitudes also exists at lower elevations. They speculated that any factor, which increased chick growth or improved feed conversion also contributed to an increased incidence of the ascites syndrome.

Research evidence points to the increased metabolic oxygen requirement of high food intake and rapid growth, as the cause of the recent marked increase in PHS in broiler chickens at both low and high altitude (Julian *et al*., 1987). PHS is related to metabolic oxygen requirement at both high and low altitude and anything that

increases O_2 requirement, increases the incidence of ascites caused by PH (Julian *et al*., 1987). Body size also affects O_2 requirement (Julian, 1993).

Pulmonary hypertension caused by increased blood flow or increased resistance to flow in the lung results in right ventricular hypertrophy, valvular insufficiency, increased venous pressure and ascites. The structure of the avian heart, with its thin – walled right ventricle and muscular right atrioventricular valve, allows pulmonary hypertension to induce heart failure quickly (Julian, 1993).

In chickens that die from ascites caused by right ventricular failure (RVF) the damage to the heart usually occurs during the first week, when percentage weight gain is the highest. These chickens grow slower and die during week four to six. It was confirmed by Julian *et al*., (1987) that broilers with ascites from RVF at processing were slightly heavier at 14 days, but because growth stops as RVF develops, they were much lighter at 47 days than their penmates.

The incidence of ascites is higher during cold weather (Huchzermeyer *et al*., 1989). Increased oxygen requirement is probably the most important factor in cold-induced PHS (Julian 1993). The temperature in the experimental unit was the same for all three treatment groups. Although the minimum temperature during some days were lower than the ideal temperature (25°C on day one and 27°C on day two), it was only for short periods. The colder temperatures on their own did not cause ascites, but in chickens on crumbles and pellets, where chickens grew the fastest, the added effect of suboptimal temperatures could have increased the incidence of ascites, due to the increase in oxygen requirements, especially during the first two weeks.

The levels of oxygen, carbon dioxide and ammonia was the same for all three treatment groups. The lowest recording for oxygen was 18,7%, the highest recording for CO_2 was 0,8% and the highest recording for NH_3 was 8,4 ppm. Although some workers have pointed to the possibility of poor ventilation causing low environmental oxygen, high carbon dioxide, or noxious fumes, causing lung damage in cold weather, there is no evidence to support this pathogenesis (Julian, 1993). Julian (1993) further reported that high levels of ammonia and stress may reduce ascites by reducing performance rather than increasing ascites.

With the environmental factors the same for all three treatment groups and the fact that the pelleting process did not influenced the incidence of ascites, it can be concluded that the percentage weight gain, especially during the first week, played the most important role in the incidence of ascites.

Sudden Death Syndrome

The incidence of SDS in the 3 treatment groups was as follows: crumbles and pellets (1,39%), ground crumbles and pellets (1,01%) and all-mash (0,51%). The incidence of SDS in chickens on crumbles and pellets was significantly (p#0,05) higher if compared to chickens on mash, but not if compared to chickens on ground crumbles and pellets (Appendix I – Table 5.5). Broilers on crumbles and pellets were 2,7 times more likely to die from SDS than broilers on mash, and broilers on ground crumbles and pellets were 2,0 times more likely to die from SDS than broilers on mash.

During the first 2 weeks the percentage weight gain in the chickens on crumbles was the highest, followed by the chickens on mash and then the chickens on ground crumbles. The percentage weight gain from week three to six was similar. The fastest growth and highest incidence of SDS were observed in the chickens on crumbles and pellets. The slowest growth was observed in the chickens on ground crumbles and pellets, but this group had a higher incidence (1,01%) of SDS when compared to the chickens on mash (0,51%), although the difference was not significant ($p>0,05$). These results agree mostly with those of Proudfoot *et al*. (1982b). They found that the overall mortality and mortality due to SDS in the crumble-pellet regimen and ground crumble-pellet regimen were almost the same.

Ononiwu *et al*. (1979) and Gardiner *et al*. (1988) both gave data indicating that chickens dying from SDS were slightly heavier than the flock average, but this correlation was not found by Riddell *et al*. (1985). This study agrees with Riddell *et al*. (1985), because in treatment group 2 and 3, 50% of the chickens that died from SDS on the days the chickens were weighed, were lighter than the average weight for those days. In treatment group 1, 63% was lighter than the average weight.

Chickens fed the ground crumble-pellet regimen had slower growth rates than chickens fed all-mash, but had mortality rates due to SDS that were higher. This provides indirect evidence that a higher incidence of SDS is not caused alone by the stress of rapid growth, but rather increased by some unidentified factor(s) involved with the crumble-pelleting process. This agrees with Proudfoot *et al*. (1982b).

The first case of SDS was diagnosed on 6 days. It gradually increased and most chickens died of SDS between 8 and 28 days.

The peak mortality due to SDS was in the third week. This correlates with the findings of Brigden and Riddell (1975) and Riddell (1993). Gardiner *et al*. (1988) found that the first deaths due to SDS occurred on day 2 and the mortality rate reached a maximum when the chickens were between 21 and 27 days of age. After this plateau it decreased gradually over the remaining time.

Jordan (1990) remarked that the peak mortality occurred from 1 to 3 weeks, coinciding with the age at which feed conversion is best. The pathogenesis of SDS is not known. The acute death with no significant lesions could be explained by ventricular fibrillation (Riddell, 1993). Greenlees *et al*. (1989) demonstrated an increased myocardial irritability in fast growing compared to slower growing male broiler chickens at 3 weeks of age, which was not observed in 6 week old birds. They suggested that the increased myocardial sensitivity is due to rapid biochemical or endocrine changes so that some birds are predisposed to SDS under certain conditions.

The etiology of SDS is unknown. It has been suggested that SDS is a metabolic disease and that genetic, nutritional and environmental factors may affect the incidence (Riddell and Orr, 1980).

Skeletal Disorders

The incidence of leg problems was the highest in the ground crumble-pellet treatment group. It was 0,96%, which is divided into infectious (0,15%) and non-infectious (0,81%) causes. The incidence in the crumble-pellet treatment group was 0,67% (0,26% infectious and 0,41% non-infectious) and 0,56% (0,31% infectious and 0,25% non-infectious) in the all-mash treatment group. There

were no significant (p>0,05) differences in leg problems between treatment groups (Appendix I – Table 5.7).

The infectious leg problems included feet- and hock joint infections, as well as femur head necrosis. Cultures were made from the infected joints and *E. coli* was confirmed. In 42,8% of cases the left leg was involved. The right leg was involved in 28,6% of cases and both legs in 28,6% of the cases.

The non-infectious leg problems were made up by VVD (89,7%) and TD (10,3%). In the chickens with VVD, both legs were involved in 57,7% of cases, the right leg in 26,9% and the left leg in 15,4% of cases. In the chickens with TD both legs were involved in 100% of cases.

Julian (1984) reported that the incidence of broiler chickens affected with VVD varies from 0,5 to 2% in normal broiler flocks, but occasionally affects 5 to 25% of male broilers in problem flocks. A 30% incidence of tibial dyschondroplasia is common in broiler chicken and turkey flocks, but many affected birds show no clinical signs (Riddell, 1993).

Riddell *et al*. (1985) reported that VVD may affect both legs, but it is often unilateral with the right leg more commonly affected than the left leg. The results of this study found that both legs were affected in most of the cases. Although numbers were small, it agreed with the finding of Riddell *et al*. (1985) that the right leg was more affected than the left.

Both Julian (1994) and Thorp (1994) speculated that VVD and TD are related to over-nutrition and rapid growth. Gordon (1994) and Julian (1994) reported an improvement in leg health when early

growth was slowed down. Riddell (1993) came to the conclusion that most of these disorders have a genetic basis, but the incidence can be influenced by nutrition and management. An association with rapid growth is apparent, but rapid growth as such is not the primary cause. This agrees with the results of this study, because the highest incidence of non-infectious leg problems was in the ground crumble-pellet regimen, which had the lowest bodyweight at 42 days.

In this study the percentage weight gain per week, did not correlate with the incidence of leg problems. The fastest weight gain was during the first two weeks in chickens on crumbles and the slowest in chickens on ground crumbles. The weight gain from week 3 to week 6 was similar.

The chickens that fed on the feed that went through the pelleting process, had a higher incidence (0,81% and 0,41%) of non-infectious leg problems when compared to the chickens on the all-mash diet (0,25%). The incidence was significantly (p #0,05) higher if chickens on ground crumbles and pellets were compared to chickens on mash, but it was not significant (p >0,05) if chickens on crumbles and pellets were compared to chickens on mash. It can therefore be concluded that the incidence of leg problems (infectious and non-infectious) was not significantly influenced by the pelleting process or the feed texture.

Septicaemia

Septicaemia was one of the more important causes of mortality. The incidence for the different groups was as follows: crumbles and pellets (0,87%), ground crumbles and pellets (0,71%) and mash

(0,56%). These differences were insignificant (p >0,05) (Appendix I – Table 5.8).

There was no correlation between final bodyweight and percentage weight gain per week and the incidence of septicaemia.

4.5 PRODUCTION EFFICIENCY FACTOR

The PEF was used to evaluate the efficiency of each group of chickens. The higher the PEF the better the performance, because the PEF takes into account the livability (mortality), liveweight, FC and age. It is a method to compare flocks within an integration. The results are given in Table 4.5.1

Table 4.5.1 Production Efficiency Factor at 42 days of age per treatment group

Treatment group	Age (days)	PEF
1	42	269,8
2	42	233,6
3	42	242,4

The PEF varied between 233,6 and 269,8 for the three treatment groups. These results compare well with the best PEF results achieved in broilers on the highveld in South Africa.

Although treatment group 1 (crumbles and pellets) had a higher mortality, the better bodyweight and FC gave it the best PEF. It was followed by treatment group 3 (mash). The ground crumbles and

pellets were the least efficient of the three rations. The better performance of the chickens on the crumbles and pellets, makes it more efficient to use crumbles and pellets for broiler production.

4.6 LIGHT INTENSITY

Table 4.6.1 Average light intensity in lux : Weekly recorded in six pens

Age (days)	Pens						Average
	I	II	III	IV	V	VI	
1 – 7	26,0	26,3	26,2	26,2	26,7	23,7	25,9
8 – 14	5,7	5,9	4,6	6,2	6,3	7,0	6,0
15 – 21	5,4	6,5	4,3	5,8	6,5	4,6	5,5
22 – 28	4,4	6,1	5,3	6,1	5,2	5,2	5,4
29 – 35	5,4	5,4	5,5	5,9	5,9	5,1	5,5
36 – 42	4,9	4,9	4,6	6,1	5,2	4,8	5,1

The average light intensity measured in six pens per week, was between 23,7 and 26,7 lux for the first week and between 4,3 and 7,0 lux from day 8 to 42. The light intensity was within acceptable standards.

4.7 OXYGEN, CARBON DIOXIDE AND AMMONIA

Table 4.7.1 O_2, CO_2, and NH_3 concentrations recorded weekly

Age (days)	O_2 (%)	CO_2 (%)	NH_3 (ppm)
8	18,9	0,8	2,4
12	18,7	0,6	2,4
17	19,3	0,4	4,8
21	19,5	0,5	8,4
26	19,4	0,2	5,9
31	20,1	0,2	7,1
36	20,9	0,1	3,6

Oxygen levels were between 18,7% at 12 days and 20,9% at 36 days. Carbon dioxide levels varied between 0,8% at 8 days and 0,1% at 36 days. Minimum ventilation during the first 3 weeks was very low to help maintain house temperature. This caused a decrease in O_2% and an increase in CO_2%. The suboptimal air quality could have had a negative influence on the growth rate, feed conversion and mortality of the broilers. The aim of the experiment was not to provide ideal conditions, but rather to mimic field conditions during winter months on the highveld. The air quality was the same for the chickens in all three treatment groups.

The ammonia was at acceptable levels of between 2,4 ppm and 8,4 ppm.

4.8 BASAL RATION

Table 4.8.1 Ration formulations

Ration	Unit	Starter	Finisher	Post finisher	Post finisher non-med
Metabolisable energy	MJ/kg	13,0	13,6	13,6	13,6
Protein	%	21,0	18,0	17,0	16,0
Fibre maximum	%	4,0	4,0	4,0	4,0
Lysine	%	1,28	1,1	1,0	0,9
Calcium	%	1,0	0,9	0,8	0,8
Available Phosphorus	%	0,5	0,45	0,4	0,4
Sodium	%	0,18	0,18	0,17	0,16
Zinc Bacitracin	g	20,0	20,0	20,0	0,0
Nitrovin	g	12,0	12,0	12,0	0,0
Sodium Monensin	ppm	100,0	100,0	100,0	0,0

4.9 SIZE OF FEED PARTICLES

Table 4.9.1. Size of feed particles, in percentage particles per size group, for treatment group 1, 2 and 3

Treatment group	Sieve size				Total particles (%)
	> 4,0 mm (%)	> 3,6 mm (%)	> 0,6 mm (%)	< 0,6 mm (%)	
1 (Crumbles)	6,0	38,0	55,0	11,0	100,0
1 (Pellets)	65,0	11,7	18,3	5,0	100,0
2	0,0	3,5	55,0	41,5	100,0
3	0,0	7,5	67,5	25,0	100,0

There was a good correlation between the bodyweight of the treatment groups and the percentage particles smaller than 0,6 mm. Treatment group1, fed on crumbles (11,0% particles <0,6 mm) and pellets (5,0% <0,6 mm) had the best bodyweight (2304,0 gram) at 42 days. Treatment group 3, fed on mash (25,0%, <0,6 mm) had a bodyweight of 2054,1 gram at 42 days. Treatment group 2 recorded the lowest bodyweight of 1993,5 gram at 42 days. They received grounded crumbles and pellets, with 41,5% of the particles smaller than 0,6 mm.

4.10 TEMPERATURE AND RELATIVE HUMIDITY

Table 4.10.1 Environmental Temperature and Relative Humidity: recorded daily over 24 hours

Age (days)	Temperature °C (min/max over 24 hours)				Relative Humidity % (min/max over 24 hours)			
	Inside		Outside		Inside		Outside	
	Minimum	Maximum	Minimum	Maximum	Minimum	Maximum	Minimum	Maximum
1	25	33	-5	19	37	55	21	85
2	27	33	-3	20	32	40	21	87
3	31	35	2	22	28	36	24	80
4	31	34	-5	20	35	48	28	85
5	31	32	6	17	36	53	42	86
6	30	32	0	11	50	62	58	87
7	29	30	-1	18	52	65	40	88
8	29	30	1	15	52	65	28	82
9	29	30	-2	15	56	66	27	86
10	28	29	-3	15	55	68	20	85
11	28	29	-6	17	31	70	17	85
12	25	28	-3	15	27	60	21	80
13	27	30	-1	14	38	69	36	85
14	25	28	-4	16	35	75	24	89
15	26	29	-3	18	28	75	23	87
16	27	29	-3	18	28	79	22	85
17	26	30	-3	19	27	73	18	88
18	26	29	-3	19	27	76	21	85
19	25	29	-1	19	27	78	22	80
20	23	27	-3	19	25	80	16	86
21	24	26	-3	20	26	65	18	85
22	23	27	-2	22	23	70	17	85
23	23	26	1	19	30	78	26	77
24	23	26	1	10	56	74	65	85
25	19	25	-2	10	43	79	46	89
26	19	23	-2	13	38	70	31	87
27	21	25	-4	15	34	76	26	87
28	20	24	-4	17	31	75	20	84
29	20	23	-3	16	36	65	27	86
30	19	23	-6	13	35	68	27	87
31	19	23	-6	14	36	74	27	90
32	15	23	-4	14	35	78	22	89
33	19	23	-5	15	35	72	23	89
34	20	23	1	15	38	75	30	85
35	17	24	0	16	36	82	25	87
36	17	24	-3	17	35	80	23	85
37	18	24	-3	16	32	86	18	87
38	17	24	-6	18	28	84	14	88
39	16	26	-5	19	30	86	18	85
40	17	25	-4	18	30	80	20	87
41	18	25	-5	18	30	80	18	86
42	19	24	1	17	47	83	27	87
Average	23,1	27,2	-2,5	16,6	35,5	70,8	26,1	85,7
Minimum	15,0	23,0	-6,0	10,0	23,0	36,0	14,0	77,0
Maximum	31,0	35,0	6,0	22,0	56,0	86,0	65,0	90,0

The complete graphs of the temperature and relative humidity, as drawn by the hygrothermographs on the inside and outside of the test house, are given in Appendix I and II and the minimum and maximum values of temperature and relative humidity as recorded daily on the inside and outside of the test house are given in Table 4.10.1.

The average minimum outside temperature was -2,5°C [-6°C – 6°C] and the average maximum outside temperature was 16,6°C [10°C – 22°C]. The average minimum outside relative humidity was 26,1% [14% - 65%] and the average maximum outside relative humidity was 85,7% [77% - 90%].

The average minimum inside relative humidity was 35,5% [23,0% - 56%] and the average maximum inside relative humidity was 70,8% [36,0% - 86,0%]. The house was not equipped to control relative humidity and it was mainly determined by the outside relative humidity and the ventilation in the house. The relative humidity was a reflection of the conditions in chicken houses during winter months on the highveld.

The average minimum inside temperature was 23,1°C [15°C – 31°C] and the average maximum inside temperature was 27,2°C [23,0°C – 35°C]. The temperature in the house was not always ideal, but the variation in temperature was acceptable and compare well with temperature variation under field conditions during winter on the highveld. The temperature and relative humidity were the same for all the chickens in the three treatment groups.

4.11 ECONOMIC EVALUATION

Table 4.11.1 Partial Farm Budgeting

	Treatment group 1 vs Treatment group 2	**Treatment group 1 vs Treatment group 3**	**Treatment group 3 vs Treatment group 2**
Additional returns Additional weight at 42 days	(R24 193,12 - R21 434,00) R2759,12	(R24 193,12 - R22 280,72) R1912,40	(R22 280,72 – R21 434,00) R846,72
Foregone returns Nil	R0,00	R0,00	R0,00
Additional costs incurred Feed cost	(R11 493,72 – R10 429,30) R1064,42	(R11 493,70 – R10 777,66) R716,04	(R10 777,66 – R10 429,30) R348,36
Costs no longer incurred Nil	R0,00	R0,00	R0,00
Net return	(R2759,12 – R1064,42) R1694,70	(R1912,40 – R716,04) R1196,36	R846,72 – R348,36) R498,36

For the partial farm budgeting the following values were used:

- Feed costs : R1380.00 per ton
- Pelleting and crumbling cost : R20.00 per ton
- Income from liveweight : R5.60 per kg, which was 80% of the meat price at the time (meat price was R7.00 per kg)

For 2000 broilers per treatment group the difference in net return between treatment group 1 (crumbles and pellets) and 2 (ground crumbles and pellets) was R1694.70 and between treatment group 1 (crumbles and pellets) and 3 (mash) it was R1196.36. In a company slaughtering 1 000 000 broilers per week the difference in net return in chickens on crumbles and pellets, compared to chickens on ground crumbles and pellets will be R847 350.00 per week and if compared to chickens on mash it will be R598 180.00 per week. These figures confirm the fact that it is much more efficient and economically viable to feed good quality crumbles and pellets to boilers.

It further emphasises the fact that feed mills must produce pelleted feed with exceptional crumble and pellet quality to ensure maximal income for broiler producers.

CHAPTER V

5. CONCLUSION

- The following conclusions can be made from this study:

  - The feeding of a combination of crumbles and pellets to male broilers, resulted in significantly (p#0,05) better growth and therefore bodyweight at 42 days of age when compared to male broilers receiving ground crumbles and pellets or an all mash diet.

  - Male broilers receiving crumbles and pellets experience significantly (p#0,05) better FC and mortality corrected FC at 42 days of age than male broilers consuming ground crumbles and pellets or only mash.

  - The better bodyweight and feed conversion in male broilers at 42 days of age receiving crumbles and pellets resulted from the bigger particle size (feed texture) of the feed, because all three treatments groups received feed of the same specification. Feed processing (pelleting) on it's own did not cause a significant improvement in 42 day bodyweights or feed conversion.

  - The percentage mortality at 42 days of age in male broilers consuming crumbles and pellets was significantly (p#0,05) higher than male broilers receiving ground crumbles and pellets or all mash. The percentage mortality in male broilers on mash was

significantly (p#0,05) lower than in the other treatment groups.

- The higher mortality in birds on crumbles and pellets was mainly due to an increased incidence of Ascites and Sudden Death Syndrome. The increased risk of death from ascites can be explained by the higher percentage weight gain during the first week and to a lesser extent during the second week, which leads to more stress on the respiratory and cardiovascular system. The higher incidence of SDS was caused by a combination of fast growth (percentage weight gain) during the first week and to a lesser degree during the second week and some unidentified factor(s) involved with the pelleting process.

- Feed texture and feed processing did not significantly influence the occurrence of leg problems and septicaemia, or any other disease (excluding SDS and Ascites) during this experiment.

- The PEF, which takes into account liveability, liveweight, FC and age, is a useful method for comparing flock results within an integration. Despite the higher mortality in male broilers receiving crumbles and pellets, their better bodyweight and FC ensured the best PEF of 269,8 at 42 days of age. The PEF of the birds receiving an all mash diet was 242,4 and on the ground crumbles and pellets it was 233,6.

- The net income from 2000 male broilers on crumbles and pellets was R1694.70 more if compared to birds on

ground crumbles and pellets and R1196.36 more if compared to birds on mash. In a company slaughtering 1 000 000 broilers per week the benefit in net return is R847 350.00 per week if a crumble and pellet diet is compared to a ground crumble and pellet diet and R598 180.00 if compared to an all mash diet.

- The better performance and net return in birds on crumbles and pellets, emphasise the importance of feed mills to produce excellent quality crumbles and pellets to help broiler producers to obtain maximum growth and feed conversion and therefore maximum income from their boilers.

- This work gives overwhelming evidence, that particle size (feed texture), plays the most important role in determining bodyweight and feed conversion in broilers. To ensure the heaviest bodyweight and the best feed conversion on any given feed specification, it is of utmost importance that broilers receive feed as intact crumbles and pellets, with minimum damage to the crumbles and pellets. It can therefore be concluded that feed texture (particle size) and not feed processing (pelleting), is the most important factor influencing broiler results, and thus income from broiler production.

❑ RECOMMENDATIONS

It can be recommended from the results of this study that the following aspects need to be investigated further:

- Ways to improve crumble and pellet quality.

- Factor(s) that cause SDS, especially the factors(s) involved with the pelleting process.

- The ideal particle size for crumbles and pellets to achieve optimum results in broiler production.

SUMMARY

THE EFFECT OF FEED PROCESSING AND FEED TEXTURE ON BODYWEIGHT, FEED CONVERSION AND MORTALITY IN MALE BROILERS

by

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A study was carried out to evaluate the effect of feed processing (pelleting) on bodyweight, feed conversion and mortality in male broilers. Pelleted feed was compared to mash feed with the same specification. In addition, the effect of feed texture (feed particle size) on bodyweight, feed conversion and mortality, by using crumbles and pellets, ground crumbles and pellets, and mash was evaluated.

Six thousand day-old Ross 788 male broiler chickens, originating from a specific broiler breeder flock (37 weeks old) were divided into

three treatment groups of 2000 birds by systematic random sampling. The experiment was an 8 x 3 block design, with 250 broilers randomly and equally assigned to each pen.

The birds were kept in a controlled environmental house and vaccinated against NCD, IB, IBD and Pneumovirus.

The experiment was carried out at 1517 m above sea level, on the Highveld of South Africa. At this altitude and together with the fact that the experiment was carried out during winter (June and July 1997), no inducing methods were necessary.

Mortalities were recorded daily and post mortems were done on all dead chickens and the cause of death recorded. Dead chickens were weighed individually and the weight recorded. Bacteriology was done on all the chickens that died from infectious causes to identify the specific bacteria. The bodyweight per pen was determined by weighing all the chickens per pen on day 0, 7, 14, 21, 28, 35 and 42 and weighing at least 20% of the chickens per pen on day 4, 11, 18, 25, 32 and 39. The feed conversion and mortality corrected feed conversion were determined on day 7, 14, 21, 28, 35 and 42. The mean live mass in kg, the percentage survivors, the feed conversion and age in days were used to calculate the production efficiency factor for each treatment group at 42 days of age.

Chickens on crumbles and pellets had the highest bodyweight (2304,0 g) at 42 days of age. They were followed by the chickens on the mash diet (2054,1 g) and the lowest bodyweight was recorded on the ground crumbles and pellets (1993,5 g). The difference in bodyweight for the three treatment groups was significant (p#0,05). The pelleting process therefore did not result in better bodyweight,

but the bodyweight were determined by the particle size of the feed (feed texture).

The percentage weight gain per week, decreased from week one to week six. During the first week and to a lesser extent during the second week, there was a big difference in percentage weight gain between treatment groups. The chickens on the crumbles and pellets grew the fastest (230,1% during week one and 159% during week two). The weight gain for the chickens on the all-mash diet was 187,7% for week one and 153,1% for week two. The slowest weight gain was in the chickens on the ground crumbles and pellets (179,7% during week one and 143,5% during week two). The weekly weight gain in the three treatment groups from week three to week six was similar.

The difference in weight gain over the first two weeks, was probably due to the difference in feed texture, because the chickens on the crumbles grew the fastest and the chickens on the ground crumbles the slowest. Eleven percent of the crumbles and 5% of the pellets were smaller than 0,6 mm. In the mash 25,0% and in the ground crumbles and pellets 41,5% of the particles were smaller than 0,6 mm. In the crumbles 44% and in the pellets 76,7% particles were greater than 3,6 mm. In the mash 7,5% and in the ground crumbles and pellets 3,5% of the particles were greater than 3,6 mm.

The best FC (1,900) and mortality corrected FC (1,852) were achieved on crumbles and pellets. It differed significantly (p#0,05) from the FC (1,946) and mortality corrected FC (1,921) of chickens on ground crumbles and pellets, as well as the FC (1,963) and mortality corrected FC (1,945) of chickens on mash. There was no significant (p>0,05) difference in the FC and mortality corrected FC in the ground crumbles and pellets, and mash rations. The pelleting

process on its own, did not significantly improve feed efficiency. Grinding of crumbles and pellets abolished the feed efficiency responses observed when the physical form was preserved. Particle size (feed texture) was therefore the most important factor determining feed efficiency.

Mortality was the highest in chickens on crumbles and pellets (6,57%), followed by 4,03% in chickens on ground crumbles and pellets and 2,85% in chickens on mash. These differences in mortality were significant (p#0,05). The higher mortality on crumbles and pellets was mainly caused by ascites (2,11%) and SDS (1,39%), which caused 3,5% of the mortality. The most important cause of mortality in the group receiving ground crumbles and pellets was SDS (1,01%).

Although the total mortality in the chickens on crumbles and pellets was the highest, the better bodyweight and FC in this group resulted in the highest PEF (269,8) at 42 days. The chickens on mash had a PEF of 242,4 and in the chickens on ground crumbles and pellets it was 233,6. The better results on crumbles and pellets are further accentuated by the net return per 2000 day-old chickens placed of R1694.70 when compared to the chickens on ground crumbles and pellets, and R1196.36 when compared to the chickens on mash.

This study therefore, showed that particle size (feed texture), played the most important role in determining bodyweight and feed efficiency in broilers. To ensure the heaviest bodyweight and most efficient feed conversion on any given feed specification, it is of utmost importance that broilers receive feed as intact crumbles and pellets, with minimum damage to the crumbles and pellets.

REFERENCES

Alfred, J.B., Fry, R.E., Jensen, L.S., and McGinnis, J., 1957. Studies with chicks on the improvement in nutritive value of feed ingredients by pelleting. *Poultry Sci.* 36:1284-1289.

Angulo, E., Bureau, J., Miquel, A., and Esteve-Garcia, E., 1993. Research note: Effect of diet density and pelleting on productive parameters of japanese quail. *Poultry Sci.* 72:607-610.

Arscott, G.H., Hulit, V.L., and Poutz, R.K., 1957. The use of barley in high-efficiency broiler rations. 3. Effect of pellets and reground pellets on growth and efficiency of feed utilization. *Poultry Sci.* 36:1388-1389.

Bayley, H.S., Summers, J.D., and Slinger, S.J., 1968. The influence of steam pelleting conditions on the nutritional value of chick diets. *Poultry Sci.* 47:931-939.

Blair, R., Newberry, R.C., and Gardiner, E.E., 1993. Effects of lighting pattern and dietary tryptophan supplementation on growth and mortality in broilers. *Poultry Sci.* 72:495-502.

Blakely, R.M., MacGregor, H.I., and Hanel, D., 1963. The effect of type of pelleting on growth and metabolizable energy from turkey rations. *Br. Poult. Sci.* 4:261-265.

Brigden, J.L., and Riddell, C., 1975. A survey of mortality in four broiler flocks in western Canada. *Can.Vet. J.* 16 :194-200.

Buys, S.B., and Barnes, P.D., 1981. Ascites in broilers. *Vet. Rec.* 108:266.

Buys, S.B., 1996. Growth related conditions in broilers. *Poultry Bulletin* August : 371-372.

Buys, S.B., 1997. *Personal Communication.*

Chambers, J.R., 1986. Heritability of crippling and acute death syndrome in sire and dam strains of broiler chickens. *Poultry Sci.* 65. Supplement 1:23.

Chambers, J.R., Gavora, J.S., and Fortin, A., 1981. Genetic changes in meat-type chickens in the past twenty years. *Can. J. Anim. Sci.* 61:555-563.

Dale, N., and Villacres, A., 1988. Relationship of two-week body weight to the incidence of ascites in broilers. *Avian Dis.* 32:555-560.

Gardiner, E.E., Hunt, J.R., and Newberry, R.C., 1988. Relationships between age, body weight and season of the year and the incidence of sudden death syndrome in male broiler chickens. *Poultry Sci.* 67:1243-1249.

Gordon, S.H., 1994. Effects of daylength and increasing daylength programmes on broiler welfare and performance. *World's Poult. Sci. J.* 50:269-282

Greenless, K.J., Eyre, P., Lee, J.C., and Larsen, C.T., 1989. *Effect of age and growth rate on myocardial irritability in broiler chickens*

(42861). Proceedings of the Society for Experimental Biology and Medicine. 190:282-285.

Hall, S.A., and Machicao, N., 1968. Myocarditis in broiler chickens reared at high altitude. *Avian Dis.* 12:75-84.

Hassanzadeh Ladmakhi, M., Buys Nadine, Dewil Erna, Rahimi, G., and Decuypere, E., 1997. The prophylactic effect of vitamin C supplementation on broiler ascites incidence and plasma thyroid hormone concentration. *Avian Pathol.* 26 : 33-44.

Havenstein, G.B., Ferket, P.R., Scheideler, S.E., and Larson, B.T., 1994. Growth, livability, and feed conversion of 1957 vs 1991 broilers when fed "typical" 1957 and 1991 broiler diets. *Poultry Sci.* 73:1785-1794.

Heywang, B.W., and Morgan, R.B., 1944. Pelleted and unpelleted all-mash diet for growing chickens. *Poultry Sci.* 23:26-30.

Hitchner, S.B., Domermuth, C.H., Purchase, H.G., and Williams, J.E., (eds.), 1980. *Isolation and Identification of Avian Pathogens.* 2nd ed. Texas, American Association of Avian Pathologists: pp 129-135.

Huchzermeyer, F.W., De Ruyck, An M.C., and Van Ark, H., 1988. Broiler pulmonary hypertension syndrome. III. Commercial broiler strains differ in their susceptibility. *Onderstepoort J. Vet. Res.* 55:5-9.

Huchzermeyer, F.W., 1989. Broiler ascites research : where are we ? *Poultry Bulletin* June : p358.

Huchzermeyer, F.W., Van Der Colf, W.J., and Guinane, P.R., 1989. Broiler ascites : increased oxygen demand with cold may explain high winter incidence. *Poultry Bulletin* September :p474 and p483.

Hussar, N., and Robblee, A.R., 1962. Effects of pelleting on the utilization of feed by the growing chicken. *Poultry Sci.* 41:1489-1493.

Jensen, L.S., and Falen., L., 1973. Effect of pelleting on the "Extra caloric" effect of dietary fat for developing turkeys. *Poultry Sci.* 52:2342-2344.

Jordan, F.T.W. (ed.), 1990. *Poultry Diseases*. 3rd ed. London, Bailliere Tindall : pp 330-353.

Julian, R.J., 1984. Valgus-varus deformity of the intertarsal joint in broiler chickens. *Can. Vet. J.* 25:254-258.

Julian, R.J., Friars, G.W., French, H., and Quinton, M., 1987. The relationship of right ventricular hypertrophy, right ventricular failure, and ascites to weight gain in broiler and roaster chickens. *Avian Dis.* 31:130-135.

Julian, R.J., and Mirsalimi, S.M. 1992. Blood oxygen concentration of fast-growing and slow-growing broiler chickens with ascites from right ventricular failure. *Avian Dis.* 36:730-732.

Julian, R.J., 1993. Ascites in poultry : a review. *Avian Pathol.* 22 : 419-454.

Julian, R.J., 1994. *Metabolic disorders and welfare of broilers.* Proc. 9th European Poultry Conference, Glasgow, U.K. 1:pp 64-69.

Lamas da Silva, J.M., Dale, N., and Luchesi, J.B., 1988. Effect of pelleted feed on the increase of ascites in broilers reared at low altitudes. *Avian Dis.* 32:376-378.

Leeson, S., and Summers, J.D., 1988. Some nutritional implications of leg problems with poultry. *Br. Vet. J.* 144:81-92.

Lilburn, M.S., 1994. Skeletal growth of commercial poultry species. *Poultry Sci.* 73:897-903.

Lott, B.D., Branton, S.L., and May, J.D., 1996. The effect of photoperiod and nutrition on ascites incidence in broilers. *Avian Dis.* 40 : 788-791.

Malan, D.D., Siebrits, F.K., Casey, M.H., Coetzer, R., and Janse Van Vuuren, P.T., 1996. The influence of growth on the development of pulmonary hypertension syndrome (ascites) and organ development in broiler chickens. *Poultry Bulletin* March : 167-171.

Marks, H.L., 1979. Growth rate and feed intake of selected and nonselected broilers. *Growth* 43:80-90.

Moran, E.E., and Summers, J.D., 1970. Factors in feed processing affecting utilization of nutrients. *Feedstuffs* 42:(Oct. 31)26-27.

Ononiwu, J.C., Thomson, R.G., Carlson, H.C., and Jullian, R.J., 1979. Studies on effect of lighting on “Sudden Death Syndrome” in broiler chickens. *Can. Vet. J.* 20:74-77.

Plavnik, I., E. Wax, Sklan, D., and Hurwitz, S., 1997. The response of broiler chickens and turkey poults to steam-pelleted diets supplemented with fat or carbohydrates. *Poultry Sci*. 76:1006-1013.

Portsmouth, J.I., and Hand, P., 1987. Trends and developments in breeding hens 1. Energy and protein utilization. *International Hatchery Practice* 2:1-10.

Proudfoot, F.G., and Hulan, H.W., 1982a. Effect of reduced feeding time using all-mash or crumble-pellet dietary regimens on broiler chicken performance, including the incidence of a acute death syndrome. *Poultry Sci*. 61 : 750-754.

Proudfoot, F.G., Hulan, H.W., and McRae, K.B., 1982b. The effect of crumbled and pelleted feed on the incidence of Sudden Death Syndrome among male chicken broilers. *Poultry Sci.* 61:1766-1768.

Qureshi, M.A., and Miller, L., 1991. Comparison of macrophage function in several commercial broiler genetic lines. *Poultry Sci.* 70:2094-2101.

Qureshi, M.A., and Havenstein, G.B., 1994. A comparison of the immune performance of a 1991 commercial broiler with a 1957 randombred strain when fed "typical" 1957 and 1991 broiler diets. *Poultry Sci.* 73:1805-1812.

Riddell, C., and Orr, J.P., 1980. Chemical studies of the blood and histological studies of the heart of broiler chickens dying from acute death syndrome. *Avian Dis.* 24 : 751-757.

Riddell, C., and Springer, R., 1985. An epizootiological study of acute death syndrome and leg weakness in broiler chickens in Western Canada. *Avian Dis.* 29:90-102.

Riddell, C., 1991. Developmental metabolic and miscellaneous disorders. In : B.W. Calnek, H.J. Barnes, C.W. Beard, W.M. Reid, and H.W. Yoder (eds). *Diseases of Poultry*. 9th edn. Ames, Iowa State University Press : pp 827-863.

Riddell, C., 1993. *Developmental and metabolic diseases of meat-type poultry.* Proc. 10th World Veterinary Poultry Association Congress, Sydney : pp 79-89.

Ross Breeders Limited, 1996. *Ross broiler management manual* : Newbridge, Midlothian, Scotland: pp 27-34.

Ross Poultry Breeders (Pty) Ltd, 1994. *Visitors information*: The Ross concept. Welverdiend Farm, Meyerton, S.A.

Sherwood, D.H., 1977. Modern broiler feeds and strains : What two decades of improvement have done. *Feedstuffs* 49:70.

Sibbald, I.R., 1977. The effect of steam pelleting on the true metabolizable energy values of poultry diets. *Poultry Sci.* 56:1686-1688.

Summers, J.D., Bentley, H.U., and Slinger, S.J., 1968. Influence of method of pelleting on utilization of energy from corn, wheat shorts and bran. *Cereal Chem.* 45:612-615.

Thorp, B.H., 1994. Skeletal disorders in the fowl : a review. *Avian Pathol.* 23:203-236.

Van der Zijpp, A.J., 1983. Breeding for immune responsiveness and disease resistance. *World's Poult. Sci. J.* 39:118-131.

Vidyadaran, M.K., King, A.S., and Kassim, H., 1990. Quantitative comparisons of lung structure of adult domestic fowl and red jungle fowl, with reference to broiler ascites. *Avian Pathol.* 19:51-58.

Zubair, A.K., and Leeson, S., 1996. Compensatory growth in the broiler chicken : a review. *World's Poult. Sci. J.* 52:189-201.

APPENDIX I

STATISTICAL ANALYSIS

Table 5.1 Bodyweight per treatment group at 42 days of age. t-Test: two-samples assuming equal variances.

	Treatment group 1	Treatment group 2
Mean	2304,000	1993,500
Variance	357,071	1231,357
Standard deviation	18,896	35,091
Observations	8,000	8,000
P(T<=t) two-tail	0,000	0,000
	Treatment group 1	**Treatment group 3**
Mean	2304,000	2054,100
Variance	357,071	608,982
Standard deviation	18,896	24,678
Observations	8,000	8,000
P(T<=t) two-tail	0,000	0,000
	Treatment group 2	**Treatment group 3**
Mean	1993,500	2054,100
Variance	1231,357	608,982
Standard deviation	35,091	24,678
Observations	8,000	8,000
P(T<=t) two-tail	0,001	0,001

Table 5.2 FC per treatment group at 42 days of age. t-Test: two-samples assuming equal variances.

	Treatment group 1	Treatment group 2
Mean	1,900	1,946
Variance	0,000	0,001
Standard deviation	0,000	0,032
Observations	8,000	8,000
P(T<=t) two-tail	0,003	0,003
	Treatment group 1	**Treatment group 3**
Mean	1,900	1,963
Variance	0,000	0,002
Standard deviation	0,000	0,045
Observations	8,000	8,000
P(T<=t) two-tail	0,002	0,002
	Treatment group 2	**Treatment group 3**
Mean	1,946	1,963
Variance	0,001	0,002
Standard deviation	0,032	0,045
Observations	8,000	8,000
P(T<=t) two-tail	0,381	0,381

Table 5.3 Mortality corrected FC per treatment group at 42 days of age. t-Test: two-samples assuming equal variances.

	Treatment group 1	Treatment group 2
Mean	1,852	1,921
Variance	0,000	0,001
Standard deviation	0,000	0,032
Observations	8,000	8,000
P(T<=t) two-tail	0,000	0,000
	Treatment group 1	**Treatment group 3**
Mean	1,852	1,945
Variance	0,000	0,001
Standard deviation	0,000	0,032
Observations	8,000	8,000
P(T<=t) two-tail	0,000	0,000
	Treatment group 2	**Treatment group 3**
Mean	1,921	1,945
Variance	0,001	0,001
Standard deviation	0,032	0,032
Observations	8,000	8,000
P(T<=t) two-tail	0,170	0,170

Table 5.4 Mortality per treatment group at 42 days of age. Statistical calculator: 2x2 tables.

	Relative risk	Confidence interval	Chi-squares	P-value
Treatment group 1 versus 2	1,60	1,22 < RR < 2,10	11,68	0,000
Treatment group 1 versus 3	2,29	1,68 < RR < 3,11	29,53	0,000
Treatment group 2 versus 3	1,48	1,05 < RR < 2,08	5,19	0,023

Table 5.5 **Incidence of Sudden Death Syndrome per treatment group from 0 - 42 days of age. Statistical calculator: 2x2 tables.**

	Relative risk	Confidence interval	Chi-squares	P-value
Treatment group 1 versus 2	1,35	0,76 < RR < 2,40	1,05	0,304
Treatment group 1 versus 3	2,70	1,31 < RR < 5,56	7,88	0,005
Treatment group 2 versus 3	2,00	0,94 < RR < 4,26	3,36	0,067

Table 5.6 **Incidence of Ascites per treatment group from 0 - 42 days of age. Statistical calculator: 2x2 tables.**

	Relative risk	Confidence interval	Chi-squares	P-value
Treatment group 1 versus 2	20,50	4,97 < RR < 84,64	35,76	0,000
Treatment group 1 versus 3	5,13	2,41 < RR < 10,90	22,50	0,000
Treatment group 2 versus 3	0,25	0,05 < RR < 1,18	3,61	0,057

Table 5.7 **Incidence of Leg Problems (infectious and non-infectious) per treatment group from 0 - 42 days of age. Statistical calculator: 2x2 tables.**

	Relative risk	Confidence interval	Chi-squares	P-value
Treatment group 1 versus 2	0,68	0,34 < RR < 1,38	1,13	0,287
Treatment group 1 versus 3	0,85	0,38 < RR < 1,88	0,17	0,682
Treatment group 2 versus 3	1,73	0,82 < RR < 3,62	2,15	0,143

Table 5.8 **Incidence of Septicaemia per treatment group from 0 - 42 days of age. Statistical calculator: 2x2 tables.**

	Relative risk	Confidence interval	Chi-squares	P-value
Treatment group 1 versus 2	1,21	0,60 < RR < 2,46	0,29	0,589
Treatment group 1 versus 3	1,55	0,73 < RR < 3,29	1,29	0,255
Treatment group 2 versus 3	1,27	0,58 < RR < 2,80	0,36	0,547

APPENDIX II

Graphs (hygrothermograph) of the temperature and relative humidity on the inside of the test house from day 0 to day 42.

APPENDIX III

Graphs (hygrothermograph) of the temperature and relative humidity on the outside of the test house from day 0 to day 42.