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THE EFFECT OF LIGHTING REGIMES AND CAPONIZATION ON MALE TURKEY PERFORMANCE, LEG WEAKNESS, PLASMA CALCIUM, SERUM $1,25\text{-}(OH)_2D_3$ AND TESTOSTERONE, AND BONE STRENGTH

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# THE EFFECT OF LIGHTING REGIMES AND CAPONIZATION ON MALE TURKEY PERFORMANCE, LEG WEAKNESS, PLASMA CALCIUM, SERUM 1,25-$(OH)_2D_3$ AND TESTOSTERONE, AND BONE STRENGTH

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## ABSTRACT

THE EFFECT OF LIGHTING REGIMES AND CAPONIZATION ON MALE TURKEY PERFORMANCE, LEG WEAKNESS, PLASMA CALCIUM, SERUM 1,25-$(OH)_2D_3$ AND TESTOSTERONE, AND BONE STRENGTH

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An experiment was designed to determine the effect of lighting treatments and caponization on turkey performance and leg weakness. Seven hundred twenty day-old Nicholas toms were used in the experiment, and observations were made on feed efficiencies (FE), body weights (BW), average daily body weight gains (ADBWG), leg weakness (LW), plasma calcium concentrations, serum 1,25-$(OH)_2D_3$ and testosterone concentrations, tibia breaking strength and maximum tensile strength. The study utilized high intensity step-up (HISU), intermittent (INT) and low intensity step-down (LISD, ie, control) lighting treatments.

Both HISU and INT lighting treatments significantly reduced LW birds without influencing FE, BW, and ADBWG. Serum testosterone and 1,25-$(OH)_2D_3$, and plasma calcium concentrations increased in the birds under HISU and INT lighting treatments. These results suggest that the decreased incidence of LW in the birds under HISU and INT lighting treatments was due to the increased serum 1,25-$(OH)_2D_3$

stimulated by the increased testosterone due to the increased light. However, the role of caponization on LW was inconclusive.

**To my wife and parents**

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## LIST OF ABBREVIATIONS

| | |
|---|---|
| ADBWG | average daily body weight gains |
| LW | leg weakness |
| BW | body weights |
| C | capons |
| Ca | calcium |
| CNTL | control |
| FE | feed efficiency |
| GLM | general linear model |
| HISU | high intensity step-up |
| INT | intermittent |
| LISD | low intensity step-down |
| M | males |
| P | phosphorus |
| SD | standard deviation |
| TSR | Tukey's studentized range test |
| TX | treatments |
| WKS | weeks |

# INTRODUCTION

Leg weakness is the term used to describe a wide variety of abnormalities affecting locomotion in poultry. A large amount of research on leg weakness in the turkey has been conducted since 1952, but this problem still exists resulting in millions of dollars in losses in the United States turkey industry annually. Laing (1976) reported 10% to 50% lameness in 16 turkey flocks studied over a 3-year period. Cost estimates on production losses due to skeletal defects in turkeys are as high as $32 million a year (Nestor et al.,1982).

It has been concluded that there is no single solution to leg weakness in poultry. The present research was concentrated on increasing the knowledge of common non-infectious skeletal deformities which have caused significant losses in the poultry industry over the past decade. When intensive turkey production was first established, leg disorders were considered a minor problem. They occurred sporadically and were generally caused by nutritional deficiencies. As the development of the intensive poultry industries progressed, genetic selection for heavy body weight

was blamed as the cause of leg weakness. Currently, the problem in turkeys is still prevalent even under apparently optimum conditions of genetics and balanced diets with enough of all known nutrients necessary for normal growth. Therefore, simple nutritional deficiencies, imbalances and genetic defects no longer suffice as the explanation for leg problems.

Management practices, such as lighting programs, appear to have an important influence. It has been consistently reported that intermittent lighting significantly reduced leg abnormalities in broilers and roasters (Buckland et al., 1973, 1974, 1976; Buckland, 1975; Wilson et al., 1984; Simons and Haye, 1985; Ketelaars et al., 1986). Similar research with turkeys by Hester and Kohl (1989) reported that an intermittent lighting program could be used in light-tight, environmentally enclosed turkey houses to reduce leg problems without affecting other production traits. However, the mechanism by which the lighting program reduces leg weakness is still unknown.

Effect of light on sex hormones and hypothesis: Photoperiod appears to be the major environmental factor which stimulates the pituitary to release the gonadotropic hormones (GH), follicle stimulating hormone (FSH) and luteinizing hormone (LH). FSH stimulates growth of seminiferous tubules and LH stimulates Leydig cells to produce testosterone and estrogen (Johnson, 1987). It has been reported that sex hormones are responsible for stimulating the 1-alpha-

hydroxylase. Deluca (1980) injected estradiol into mature female birds to a level of circulating estradiol similar to a laying female bird resulting in a rapid increase of the 1-alpha-hydroxylase and a rapid decrease of the 24-hydroxylase. This was followed by an increased serum calcium concentration. My hypothesis is that special light treatments might reduce leg weakness through regulation of calcium homeostasis by influencing biosynthesis of sex hormones and 1,25-$(OH)_2D_3$. The objectives of the present study were: (1) to investigate the effects of lighting programs on feed efficiency, growth rate and leg weakness; (2) to determine if sex hormones affect leg weakness; and (3) to observe if there were differences in plasma Ca and serum 1,25-$(OH)_2D_3$ due to the lighting programs.

# LITERATURE REVIEW

## CAUSES OF LEG WEAKNESS

### GENETIC

Leg weakness in turkeys is most commonly observed between 8 and 20 weeks of age (Norris, 1971; Sanger et al., 1974). It occurs primarily in toms although hens can also be affected (Glass,1971; Buffington et al., 1975; Laing, 1976). A number of factors have been cited as causes of or associated with leg disorders in the turkey including genetic factors (Nestor, 1984; Nestor et al., 1987), nutritional deficiencies (Sauver, 1984) and poor management such as excessive contamination of litter with bacteria (Veltmann and Jensen, 1979), improper lighting (Buckland et al., 1976; Hester and Kohl, 1989) and lack of exercise (Haye et al., 1978). Direct selection for increased body weight and breast muscles of the turkey has resulted in increased total body weight and a disproportionate deposition of breast muscles when compared to leg muscles (Marsden, 1940; Miller, 1968; Clayton et al., 1978). The relative amount of leg muscle also declines with

age as the bird gets heavier (Harshaw et al., 1940).

Skeletal changes are similar to those observed in muscles of aging turkeys (Clayton et al., 1978). It was reported that a biologically incompatible combination of increases in total body weight in conjunction with relatively less support resulted in pressure on the legs capable of magnifying the effects responsible for various types of leg problems (Nestor and Emmerson, 1990).

Several studies supported the above results. Riddell (1980) surveyed five commercial flocks of Nicholas male turkeys and found that long bone distortion was the most consistent and frequent cause of leg problems. Nestor (1984) reported that genetic increases in body weight, in a long-term growth-selected line of turkeys, were associated with increased leg problems. Body weight of both sexes consistently increased with each generation. Walking ability, as measured by a rating system of 1 (good legs) to 5 (poor legs), was poorer in the growth line and was generally declining from generation 14 to the 17th or 18th generation of selection for increased body weight.

However, some studies are not in agreement with the above results. Buffington et al. (1973) found that the lightest bird of each sex for a medium weight strain of turkeys was just as likely as the heaviest bird to have leg and foot abnormalities. It is possible that body weight does not have as large an influence on leg problems in the medium

weight strains as in larger strains. Dietary reduction in body weight gains did not influence the incidence of leg problems in a study by Adams and Stadelman (1978). Perhaps the relative amount of leg bones and muscle have not changed with the observed reduction of body weight in the medium birds. Cook et al. (1984) observed that artificial weight loading of turkeys did not increase the frequency of leg abnormalities. However, the experiment was carried out to only 4 weeks of age and body weight would not be expected to have a large influence at this age.

Nestor and Emmerson (1990) have been trying to reduce leg weakness in turkeys by increasing shank width through genetic selection. Shank width is a trait that is easily measured on the live bird, and consequently is readily adaptable to mass selection. Shank width is highly heritable and appears to be strongly correlated with both bone shank weight and the weight of the tibiotarsal and femur bones. In turkeys, selection for increased shank width leads to a general increase in the weight of individual leg bones and an increase in total leg bone mass. Thus, selection for shank width may provide a means to circumvent losses of walking ability in lines selected for increased body weight.

An alternative approach to increasing leg support would be to select for increased leg muscle development. Unfortunately it is difficult to find live bird measures which are closely related to leg muscle mass. Therefore, mass

selection for increased leg muscle is not currently possible.

Family selection for leg muscle mass in turkeys has proven to be inefficient in increasing leg muscle due to a low intensity of selection and the poor reproductive performance of large-bodied turkeys (Nestor and Emmerson, 1990). Technological advances allowing for the measurement of leg muscle development on the live bird are necessary to allow for effective direct selection for leg muscle mass.

## NUTRITIONAL

During the past several decades, the role of nutrition in the development of avian leg deformities has been a topic of intensive study. It is well established that many primary nutritional deficiencies can cause leg weakness. These include several mineral (calcium, phosphorus, manganese, and zinc) and several vitamin ($D_3$, A, E, $B_{12}$, niacin, folacin, biotin, and choline) deficiencies. Vitamin $D_3$, calcium and phosphorus deficiencies, however, are the most important causes of leg weakness.

### VITAMIN $D_3$

Metabolism: Vitamin $D_3$ activity in birds is from two sources: one from photobiogenesis and the other from the diet. Photobiogenesis of vitamin $D_3$ occurs in the skin. In the

presence of light (wavelength of 210-310 nm), pro-vitamin $D_3$ (7-dehydrocholesterol) in the skin is converted to pre-vitamin $D_3$ and then the pre-vitamin $D_3$ is slowly converted to vitamin $D_3$ in the skin. Vitamin $D_3$ from the skin and diet is absorbed into the circulatory system by the blood transport protein (vitamin D-binding protein) and transported to the liver where it is hydroxylated to 25(OH)$D_3$ by 25-hydroxylase. The 25(OH)$D_3$ is then released into circulation and transported to the kidney where it is further hydroxylated to the active form 1,25$(OH)_2D_3$ by 1-alpha-hydroxylase and to 24,25$(OH)_2D_3$ by 24-hydroxylase (Collins et al., 1991). It has been shown that 1,25$(OH)_2D_3$ has more vitamin $D_3$ activity than 24,25$(OH)_2D_3$.

1,25-$(OH)_2D_3$ and mineral homeostasis: The classical target tissues for 1,25$(OH)_2D_3$ are those tissues that have been found to be directly involved in the regulation of mineral homeostasis. Together with parathyroid hormone (PTH), 1,25$(OH)_2D_3$ exerts its action on the intestine, kidney and parathyroid gland.

The 1,25$(OH)_2D_3$ maintains serum calcium and phosphorus levels and provides mineral for bone formation mainly through its actions in the intestine. One of the best characterized effects of 1,25$(OH)_2D_3$ is the stimulation of intestine lumen-to-plasma flux of calcium and phosphorus (Norman et al., 1982; Haussler et al., 1977; DeLuca et al., 1983). 1,25$(OH)_2D_3$ treatment is known to alter the biochemical and morphological

characteristics of the intestinal cells. The size of the villus and microvilli increases upon $1,25(OH)_2D_3$ treatment. The brush border undergoes noticeable alterations of structure and composition of cell surface proteins and lipids, occurring in a time frame corresponding to the increase in $Ca^{2+}$ transport mediated by $1,25(OH)_2D_3$ (McCarthy et al., 1984).

The kidney is the major site of synthesis of $1,25(OH)_2D_3$ and of several other hydroxylated vitamin D derivatives. The most important effect $1,25(OH)_2D_3$ has on the kidney is the inhibition of $25(OH)D_3$-1-alpha-hydroxylase activity which results in a decrease in the synthesis of $1,25(OH)_2D_3$ (Clements et al., 1987; Henry and Norman, 1984). Simultaneously, the activity of the $25(OH)D_3$-24-hydroxylase is stimulated.

Although vitamin D is a powerful antirachitic agent, its primary effect on bone is the stimulation of bone resorption leading to an increase in serum calcium and phosphorus levels (Underwood and Deluca, 1984). With even slight decreases in serum calcium levels, PTH is synthesized, and it stimulates the synthesis of $1,25(OH)_2D_3$ in the kidney. Both of these hormones stimulate bone resorption. Maintaining constant levels of calcium in the blood is crucial, whether calcium is available from the diet or not. Therefore, the ability to release calcium from its largest body store, the bone, is vital. Bone is a dynamic tissue which is constantly being remodeled. Under normal physiological conditions, bone formation and bone resorption are tightly balanced. The

stimulation of bone growth and mineralization by $1,25(OH)_2D_3$ appears to be a indirect effect due to the provision of minerals for bone matrix incorporation through an increase of intestinal absorption of calcium and phosphorus (Underwood and Deluca, 1984).

Requirements of vitamin $D_3$: Vitamin $D_3$ requirements have been studied by numerous workers. Some have reported the requirement of starting poults to be approximately 800-900 I.U./kg of diet (Olsson, 1950; NRC, 1984). Using several criteria of measurement (relative bone mineral mass, bone ash determination and breaking strength of the bones), Cantor et al. (1980) compared levels of 0, 300, 900 and 1200 I.U. vitamin $D_3$ per kg of diet in poult diet to 4 weeks of age. All measurements showed improved calcification of the bones with increasing levels up to 1200 I.U. vitamin $D_3$/kg of diet.

Studies conducted by Stevens et al. (1984) indicate that a level of 2700 I.U. vitamin $D_3$/kg of diet provides an important margin of safety both in the diet of the breeding turkey hens and in the diet of the starting poults. No evidence has been obtained of any detrimental effects from this level of vitamin $D_3$.

Care must be taken in the selection of the source of the vitamin $D_3$ supplement to be used in diets for turkeys. An early study by McGinnis and Carver (1946) showed wide variations in the activities of cod liver oil, salmon oil and

irradiated animal sterol as vitamin $D_3$ sources for poults. In that study, it concluded that the poult, like the chick, could not make use of irradiated ergosterol (vitamin $D_2$).

Some commercial vitamin $D_3$ supplements do not contain the guaranteed level of vitamin D activity for young poults. Yang et al. (1973) studied 22 commercial vitamin $D_3$ supplements and they found 9 supplements to have much lower biopotencies than their stated values.

In view of all of these factors which may alter the vitamin D nutrition of turkeys, generous margins of safety have been used by Scott (1987) in setting the vitamin $D_3$ allowances, especially for breeders and for starting poults (Table 1).

Table 1. Vitamin $D_3$ Allowances for Turkeys (I.U./kg diet)

| | Starting | Growing | Finishing | Breeding |
|---|---|---|---|---|
| $D_3$ | 3600 | 1800 | 1000 | 3000 |

(Scott, 1987)

Symptoms of vitamin D deficiency: Turkeys with vitamin D deficiency show leg weakness (rubbery legs in very young poults), poor growth, awkwardness of gait, rubbery beaks, ruffled feathers, and increased mortality. The tibia and femur show markedly reduced bone ash. Serum vitamin $D_3$ , calcium and phosphorus are lower than normal levels (Scott, 1987).

## CALCIUM (Ca) AND PHOSPHORUS (P)

Function of calcium and phosphorus: Calcium and phosphorus are closely associated in metabolism, particularly in the formation of bone (Scott et al., 1976). In the growing bird, the major portion of the calcium in the diet is used for bone formation. Calcium constitutes almost one-third of the weight of the fat-free dried bone. In the mature female bird, the majority calcium is used for eggshells. Calcium is also essential for clotting of the blood, is required along with sodium and potassium for the normal beating of the heart, and is involved in the maintenance of acid-base equilibrium.

In addition to being of major importance as a constituent of bone, phosphorus is also an essential component of organic compounds involved in almost every aspect of metabolism. Phosphorus plays an important part in muscle, energy metabolism, carbohydrate, amino acid and fat metabolism, nervous tissue metabolism, normal blood chemistry, skeletal growth and transport of fatty acids and other lipids. (Scott et al., 1976).

Requirements of calcium and phosphorus: Wilgus (1931) was first to establish the quantitative limit for Ca and P for normal bone formation in chicks. He showed that the diet must contain not only minimum levels of the two elements, but also an optimum ratio of Ca:P. He also reported that the

minimum available P requirement was about 0.5% of the total diet; and the Ca:P ratios needed for normal growth in chicks varied between 1.0:1.0 and 2.2:1.0. The 2.5:1.0 ratio was borderline while a ratio of 3.3:1.0 appeared to be disastrous, producing rickets and other leg abnormalities.

The Ca sources presently used include fish meal, bone meal, dicalcium phosphate, oystershell and limestone, while soybean meal, monocalcium phosphate, dicalcium phosphate, sodium phosphate and potassium phosphate are used for P sources.

The requirements of Ca and P for turkeys for different ages are shown in table 2 (Nutrient Requirements for Poultry, 1984).

**Table 2.** Ca and P requirements of turkeys (% of diet)

| | Age (weeks) | | | | | |
|---|---|---|---|---|---|---|
| M | 0-4 | 4-8 | 8-12 | 12-16 | 16-20 | 20-24 |
| F | 0-4 | 4-8 | 8-11 | 11-14 | 14-17 | 17-20 |
| Ca | 1.20 | 1.00 | 0.85 | 0.75 | 0.65 | 0.55 |
| P | 0.60 | 0.50 | 0.42 | 0.38 | 0.32 | 0.2 |

(Nutrient Requirements for Poultry, 1984)

Ca and P deficiencies: Ca deficiency causes cage layer fatigue (CLF). CLF is a type of osteoporosis characterized by withdrawal of calcium phosphate, not only from medullary bone but also from cortical bone, particularly of the long bones of the legs. The bone becomes so thin that it easily fractures,

or so much mineral is removed from the bones that they no longer are capable of supporting the weight of the hens. This disease occurs almost entirely in hens that are confined to cages.

Severe P deficiency or lack of availability of P in the diet results in early loss of appetite, weakness and death within a period of 10 to 12 days. A less severe P deficiency causes rickets and growth failure, but apparently does not reduce the P level of the blood to an extent that interferes with the availability of P for the formation of high energy phosphates, DNA, RNA and enzymes. Even during starvation, catabolism of bone releases sufficient P for the organic phosphates needed by the body and also results in a continuous loss of P into the urine (Scott et al., 1976).

## ENVIRONMENTAL AND MANAGERIAL

**LIGHTING:** Lighting changes which can be considered in management of poultry include color (wavelength), light intensity and photoperiod. An increased incidence of tibial dyschondroplasia (TD) and a reduced incidence of valgus-varus deformation (VVD) occurred in roasters reared under fluorescent as opposed to incandescent light, but no significant effect was found on body weight (Hulan and Proudfoot, 1987). A high intensity step-up light program in turkeys resulted in a decrease in leg abnormalities when

compared with a low intensity step down light program (Hester et al., 1983). In general, single daily shorter photoperiods have reduced leg weakness when compared with continual light, but this has been associated with reduced growth. Intermittent lighting programs characterized by repeated light and dark periods during a 24 hour day have reduced leg abnormalities, but with equal or superior growth rates in growing broilers. Classen and Riddell (1989) reported that increasing the photoperiod length during the growing period of chicks consistently reduced leg problems, mainly VVD, while maintaining similar or slightly superior growth rate and feed conversions when compared with continual light. In considering mechanisms whereby some of the lighting programs mentioned above reduce the incidence of leg problems, Riddell (1990) indicated that it was important to emphasize that it had been known for some time that continual light might induce eye lesions in poultry, enlarged adrenal glands and a changed response to stressors. He suggested that circadian rhythms may be important in birds' health and may influence leg problems. Hester et al. (1983) employed a high intensity step-up lighting program to reduce leg problems in turkeys and reported that these birds had increased plasma androgens and testicular weights.

**FEED RESTRICTION:** Feed restriction can be quantitative or qualitative. Quantitative feed restriction can be

accomplished by physical restriction or by dilution. Severe physical feed restriction will reduce the incidence of VVD (Riddell, 1983) and TD (Wise and Nott, 1975). Feed restriction by dilution will also reduce the incidence of TD (Riddell, 1975). Qualitative feed restriction by either decreasing the energy or protein level at an early age will decrease the incidence of VVD (Rizk et al., 1980) and TD (Poulos et al., 1978) in broiler chickens. Feed restriction will also decrease leg weakness in turkeys (Adams and Stadelman, 1978; Ferket and Sell, 1989). In most of the above studies, the feed restriction also significantly reduced body weight. This may suggest that such procedures are not practical. However, if feed restriction only happens at an early age, the weight lost during the early feed restriction can be compensated by later growth. Complete compensation for weight lost during an early feed restriction period of 6 days can occur by 8 weeks of age in broiler chickens (Plavnik and Hurwits, 1985).

The most obvious mechanism by which feed restriction reduces the incidence of skeletal problems would appear to be a reduction of growth rate.

However, Adams et al. (1993) studied the effects of amino acid restriction during starter and grower periods on subsequent performance and incidence of leg disorders in male large white turkeys and reported that there was no significant effect of dietary amino acid level and time of restriction on incidence or severity of leg disorders of large white male

turkeys at 18 weeks of age.

**CAGING:** A higher incidence of VVD occurs in birds raised in cages compared with the ones raised in floor pens (Akpobome and Fanguy, 1989). In contradiction, a lower incidence of TD may occur in broiler chickens raised in cages compared with broiler chickens raised in floor pens (Simons, 1986).

Birds activity is probably considerably less in cages. Exercise is needed for proper bone development (Rodenhoff and Dammrich, 1973). Strength of bones of broiler chickens raised in cages may be less than those raised on litter, particularly in hot weather (Siegel et al., 1973).

**LITTER:** Abbott et al. (1969) observed that the occurrence of footpad dermatitis in turkey poults was related to the wetness and crustiness of the litter in the brooder house. At 6 weeks of age the incidence of footpad dermatitis was between 20 and 40% for birds in the North side of the house which had damp and crusty litter, while the incidence was only 10% or less in pens in the South side of the house where the pens were much drier. Following a period of 2 to 3 weeks with no rain, all of the pens became dry and the occurrence of footpad dermatitis almost disappeared. Charles and Fortune (1977) and Harms and Simpson (1977) also observed that litter conditions affected the incidence of footpad dermatitis in turkey poults. The latter workers observed a

high incidence of the dermatitis in poults kept on wet litter (produced by sprinkling water) beginning when the poults were 5 days of age. Adding supplemental biotin to the diet failed to reduce the increased incidence of dermatitis associated with the wet litter. Martland (1985) also reported that wet litter caused severe ulceration of the skin of broiler chickens, and reversion of the litter condition resulted in rapid healing of the lesions.

## CLASSIFICATION AND CLINICAL SIGNS OF LEG WEAKNESS

### INFECTIOUS DISEASES

**AVIAN MYCOPLASMOSIS:** This is a virus-like disease caused by Mycoplasma synoviae (MS). MS invades the synovial membranes resulting in swollen joints, tendon sheaths and footpads. Pomeroy et al. (1971) conducted three experiments in turkeys and reported that MS might cause a moderate to severe airsacculitis, but had a tendency to resolve after 4 to 6 weeks post infection, however, synovitis became quite apparent as the flock reached 12 weeks of age. Another experiment was conducted by Olson (1971) at West Virginia University. Five out of the 6 turkeys inoculated into the foot pad with MS developed swelling within 5 days as a result of the MS inoculation. It was indicated that MS caused a severe arthritis and lameness in turkeys.

**OSTEOMYELITIS:** This disease is causes by Escherichia coli. It is characterized by lameness. Poss and Johnson (1971) indicated that it was a chronic disease that normally occurred from 10 weeks of age up to market time. It was one of the leading causes of lameness and mortality of growing turkeys (Bagley, 1971).

**STAPHYLOCOCCOSIS:** Staphylococcosis is an acute or chronic infectious noncontagious disease of poultry caused by Staphylococcus aureus. It is characterized in the acute form by septicemia, in the chronic form by arthritis or bumblefoot or both (Schwartz, 1988). It occurs in all fowl, especially turkeys, chickens, gamebirds, and water fowl. Birds 4 to 6 weeks of age are extremely susceptible. This disease has been reported periodically for 45 years in domestic poultry. Clinical signs include swollen joints , swollen foot-pads, lameness and breast blisters.

## NONINFECTIOUS DISEASES

**CHONDRODYSTROPHY (OSTEOCHONDRODYSTROPHY, PEROSIS):** Manganese, choline, niacin, pyridoxine, zinc, biotin and folic acid deficiencies result in growth plate abnormalities that can cause enlargement and deformity of the bones of the tibiotarsal joint with secondary displacement of the gastrocnemius tendon.

Caskey et al. (1939) indicated that manganese deficiency produced a reduced bone ash and chondrodystrophy characterized by shortening of the long bones.

Perosis due to deficiency of choline has been described in turkeys (Jukes, 1940) and in chickens (Hogan et al., 1941). In both species, the leg bones were shortened and thickened while the hock joints were distorted. Wise et al. (1973) described histopathological changes in the proximal growth cartilages of the humerus, tibiotarsus and tarsometatarsus of choline deficient poults.

Gries and Scott (1972) reported a failure to grow, ataxia and death in chicks fed low levels of pyridoxine (vitamin $B_6$). Severe perosis was developed by two weeks of age in these birds.

Young et al. (1958) observed that a diet deficient in zinc, but containing adequate levels of all other known nutrients produced symptomatic perosis.

Dobson (1970) described the lesions produced by a biotin deficiency in poults at three weeks of age as a bowing of the legs associated with dermatitis. Patrick et al. (1942) found that the incorporation of biotin into the diet of turkeys could relieve both the symptoms of perosis and foot pad dermatitis. Jensen and Martinson (1969) reported that there was more leg weakness in turkeys at 18, 20 and 24 weeks of age when low biotin rations were fed during the first few weeks of life.

Clinical perosis was also reported in chicks fed adequate levels of all known nutrients, but only low levels of folic acid (Daniel et al., 1946).

In addition, heritable chondrodystrophy was reported in broilers (Asmundoson, 1942) and in turkeys (Asmundoson, 1944). Somes (1969) reported chondrodystrophy due to an autosomal recessive gene. Chicks were normal at hatching, but an enlargement of the hock joint and bending of the tibia and metatarsus outwards occurred between 3 and 5 weeks of age.

Leach et al. (1977), in efforts to characterize the chemical and histological changes associated with inherited chondrodystrophy, found that the cartilage of affected turkey embryos contained less than 50% of the galactosamine-mucopolysaccharides present in normal cartilage and that the cartilage had considerably less extracellular matrix.

**RICKETS (OSTEOMALACIA, SOFT BONES):** Deficiencies of vitamin $D_3$, Ca, P, or Ca/P imbalance result in poorly mineralized bones in young birds. The name derived from the old English word "wrikken" meaning to bend or twist (Scott et al., 1982). Lameness occurs when the ends of the long bones become enlarged (Wise, 1975). Bones are soft or rubbery. There may be beading or infolding of the ribs.

Brenes (1971) fed poults with diets varying in calcium, phosphorus, and vitamin $D_3$. Rickets was first observed at 5 days with a low phosphorus diet, at 6 days with a high

phosphorus diet, at 7 days with a low calcium diet, at 10 and 11 days in poults fed no or a very low level of vitamin $D_3$. He also reported that birds fed these diets to 24 days of age and then fed normal diets recovered at different rates. All turkeys appeared normal at 6 weeks of age except those birds previously fed low or high phosphorus diets which developed a high incidence of leg disorders from 10 to 20 weeks of age.

Rickets is generally associated with a nutritional deficiency (Jubb and Kennedy, 1970), although inherited forms of rickets have been described.

Austic et al. (1977) conducted a study involving a sex linked dwarf strain of chickens. Results indicated that when optimal dietary levels of vitamin $D_3$, calcium and phosphorus were provided, the maximal level of bone mineralization was significantly lower for dwarfs when compared to controls. It was consequently suggested that the apparent susceptibility of the dwarf strain to rickets was the result of genetic alterations in the rate of deposition of bone minerals.

Kramer and Waibel (1978) reported that when turkey breeder hens received 2400IU vitamin $D_3$/kg in their diet, none of their progeny raised on a vitamin $D_3$-deficient diet had leg problems at two weeks of age. However, when hens received 0 or 300 IU/kg of vitamin $D_3$ in their diet, a 40 to 60% occurrence of rickets resulted in their progeny.

**TIBIAL DYSCHONDROPLASIA (TD):** TD is a skeletal disorder

prevalent in commercial flocks of both turkeys and broilers. It is characterized by a widened, unmineralized, unvascularized plug of abnormal cartilage adjacent to the epiphyseal growth plate (Leach and Nesheim, 1965). The lesion also occurs in the tarsometatarsus, although not to the extent observed in the tibia (Siller, 1970). The etiology is multifactorial. Nutritional factors include metabolic acidosis, fusarochromanone, copper deficiency, excesses of cystine, homocystine and histidine. Non-nutritional factors have been identified which increase the incidence and severity of the lesion, including genetic selection (Leach and Nesheim, 1965; Nestor et al., 1985), infectious diseases (Cook et al., 1984), caponization (Johnson and Rendano 1984), lighting program (Hester et al., 1983, 1985, 1986; Buckland et al., 1973, 1976; Simons, 1982; Simons and Haye, 1985; Wilson et al., 1984), and mycotoxins and thiram (Veltmann et al., 1985; Wu, 1990, Wu et al. 1991). The clinical signs described in most of the reports include reluctance of birds to move, abnormal posture, stilted gait, and bilateral swelling of the femoral-tibial joints, often associated with anterior lateral bowing of the tibia. This disorder is similar to a generalized cartilage defect called osteochondrosis which occurs in rapidly-growing domestic animals, such as horses (Rejino and Stromberg, 1978) and pigs (Reiland,1978; Hilley, 1982). TD also bears a number of similarities to metaphyseal chondroplastic defect in endochondral ossification occurring

in humans (Rimoin et al., 1974). The incidence of the TD can be as high as 70-90% under experimental conditions (Edwards, 1989). Although the lesion regresses with advancing age, the tibiotarsus and other affected bones often remain severely deformed in the afflicted birds (Siller,1970). Clinically, the affected birds become lame and reluctant to move. Further, major economic loss results from bone fractures during processing.

**CAGE LAYER FATIGUE (OSTEOPOROSIS, OSTEOPENIA, FRAGILE BONES, BRITTLE BONE DISEASE):** Cage layer fatigue (CLF) is the most significant skeletal disease of the caged layer in production (American Association of Avian Pathologists, 1987). CLF is also considered the primary cause of fractures and breakage at the processing plant in spent fowl (Riddell, 1989). In the mid-fifties, the term CLF was first used by Couch (1955) to describe a combination of leg weakness and acute deaths.

The demands to produce eggs in layers put a tremendous stress on the calcium homeostatic mechanisms of the birds. The layer hen puts 2.3 grams of calcium into each egg shell and another 25 milligrams into the yolk (Etches, 1987). Over a 70 week period of lay, the hen may utilize greater than 580 grams of calcium for egg shell formation. Egg shell formation occurs for approximately 20 hours prior to ova position, primarily in the dark phase when the hen is not feeding. The demand for

calcium during egg shell formation is intermittent reaching a peak midway through egg shell formation. Approximately 50-60% of calcium consumed is absorbed (Hurwitz and Bar, 1969). The amount of calcium available for absorption during the dark phase depends on both the type and amount of calcium consumed during the light phase. Large particle calcium , such as oyster shell, persists longer in the intestinal tract providing more calcium for the dark phase.

The plasma concentration of calcium is usually maintained between 20-30mg/dl during the ovulation cycle and the period of egg shell calcification even with the high demand and turnover of calcium for egg shell formation (Etches, 1987; Wideman, 1987). Calcium is transferred from the blood to the egg shell at an approximate rate of 100-150 mg/hour (Soares, 1984). The plasma calcium would be reduced to zero within 15-30 minutes if alternative sources of calcium were not available (Etches, 1987). A specialized woven bone (medullary bone) formed under the influence of estrogen is presumed to provide the necessary calcium to main calcium homeostasis (Simkiss, 1967). It is estimated that 30-40% of egg shell calcium comes from bone (Mueller et al., 1977). The presumption is based on several general observations: the turnover rate of medullary bone is twice that of cortical bone (Hurwitz, 1965), medullary bone is maintained at the expense of cortical bone during periods of calcium deprivation (Simkiss, 1967) and the mass of medullary bone is greatest

immediately prior to the onset of egg shell calcification (Candlish, 1981). In addition, the osteoclast number and resorption surface increase in medullary bone during egg shell calcification (Miller, 1981) and acid phosphatase activity is increased in the plasma (Taylor et al., 1965).

The etiology of CLF has been considered to be primarily nutritional (Riddell et al., 1968) although both strains of birds and housing affect the incidence (Francis, 1957). Frequently, birds in peak production are reported to develop this problem. The lesions have been described as an osteoporosis of the cortical bone (Bell and Siller, 1962) or osteomalacia involving the nonweight bearing medullary bone (Antillon et al., 1988).

**SPONDYLOLISTHESIS (KINKY-BACK):** Kinky-back is ventral dislocation of the anterior end of the 6th thoracic vertebrae with over-riding by the 7th to cause pinching of the spinal cord. It is a heritable disease and mainly occurs in chickens (Julian, 1990).

**LONG BONE DISTORTION:** Long bone distortion is a general term used for a variety of bone deformities in turkeys and chickens. It is characterized by crooked, bent long bones with no shortening. It includes such things as asymmetry of the condyles and bowing, angular deformity, and backward bending of the tibia or metatarsus. Bone strength is normal. This

disease is normally caused by genetic factors and/or is secondary to tibia dyschondroplasia (Julian, 1990).

**ROTATED TIBIA:** Rotated tibia is an outward (lateral) rotation (torsion) of the distal part of the shaft of the tibia through its length. The bone remains straight and strong. It causes up to 5% of lameness in turkeys, and about 1% in broiler chickens. The etiology is unknown (Julian, 1990).

**VALGUS-VARUS DEFORMITY (ANGULAR BONE DEFORMITY, TWISTED LEGS):** Valgus-varus deformity is a lateral or medial deviation of the distal tibiotarsus with a corresponding deviation of the metatarsus and secondary displacement and sometimes complete slippage of the gastrocnemius tendon. It occurs in meat type poultry from a few days of age to processing. Growth plates are normal in the distal tibiotarsus, but the proximal metatarsus may be enlarged. Intertarsal ligaments become stretched and the joint is slack. Supplementing a diet with pyridoxine reduced the incidence of twisted legs in broiler chickens (Cope et la., 1979).

# MATERIALS AND METHODS

## EXPERIMENTAL DESIGN

A total of 720 day-old Nicholas toms were randomly distributed among 24 identical floor pens measuring 4.6 m x 3.0 m, resulting in 30 poults per pen. Five poults were randomly selected from each pen and one leg was banded for identification for blood samples. Each pen was equipped with a gas brooder for heating, a brooder guard for keeping poults close to the source of feed, heat and water, a flat tray for feed, a vacuum fount-type waterer and a hanging bell-type waterer for drinking. Each pen was bedded with wood shavings as litter. The litter was covered with cheesecloth for the first three days of the experiment to prevent birds from eating the wood shavings. It was then removed for the remainder of the experiment. Two cylindrical feeders replaced the feed tray after the first week and the fount-type warterer was removed at the same time. The heights of the feeder and the waterer were adjusted periodically with the growth of the turkeys. Wood shavings were added as needed to keep the litter in good condition. Each pen initially received 22 kg of litter

resulting in a average depth of 7-8cm. Each room was equipped with a fan for ventilation and temperature control.

Turkeys were randomly assigned to each of three lighting programs for a 17-week period with 8 replicates for each treatment. The three lighting programs are shown in Table 3.

All the diets were formulated to contain a constant apparent metabolizable energy, crude protein, and Ca:P ratio. A starter, two grower, and two finisher rations were fed beginning at 0, 3, 7, 12, and 16 weeks of age. Rations were formulated to meet or exceed all known turkey nutrient requirements. Table 4 shows the calculated analysis of the commercial diets used in this experiment.

Feed and water were provided ad libitum throughout the entire study. Ambient temperature was maintained at approximately 90 to 95 $^{0}$F from 0 to 2 weeks of age, and subsequent decrease of 5 $^{0}$F per week occurred when the turkeys were 3 weeks of age. Thereafter, temperature fluctuated depending on outside environmental conditions, but attempts were made to maintain from 60 to 65 $^{0}$F for the remainder of the experiment.

Thirty birds were caponized at 14 days of age in each lighting program. Birds were checked twice every day for leg abnormalities and deaths. Records of leg abnormalities, mortality and feed consumption were maintained through the study.

**Table 3.** Three Lighting Treatments Used in the Experiment

| Light program | Age (day) | Light (hour) | Intensity (lux) |
|---|---|---|---|
| High Intensity Step-up | 1-8 | 24 | 10 |
| | 9-54 | 9 | 2.5 |
| | 55-61 | 10 | 20 |
| | 62-68 | 10.5 | 20 |
| | 69-75 | 11 | 20 |
| | 76-82 | 11.5 | 20 |
| | 83-89 | 12 | 20 |
| | 90-96 | 12.5 | 20 |
| | 97-103 | 13 | 20 |
| | 104-110 | 13.5 | 20 |
| | 111-119 | 14 | 20 |
| Intermittent | 1-8 | 24 | 10 |
| | 9-54 | 4(dark)x2(light) | 2.5 |
| | 55-119 | 4(dark)x2(light) | 20 |
| Low Intensity Step-down (control) | 1-8 | 24 | 10 |
| | 9 | 23 | 2.5 |
| | 10 | 22 | 2.5 |
| | 11 | 21 | 2.5 |
| | 12 | 20 | 2.5 |
| | 13 | 19 | 2.5 |
| | 14 | 18 | 2.5 |
| | 15 | 17 | 2.5 |
| | 16 | 16 | 2.5 |
| | 17-119 | 15 | 2.5 |

**Table 4.** Calculated Analysis of Turkey Commercial Rations Used in the Experiment

| | Starter 0-3wks | Grower1 3-7wks | Grower2 7-12wks | Finisher1 12-16wks | Finisher2 16-17wks |
|---|---|---|---|---|---|
| ME, Cal/lb | 1300 | 1350 | 1400 | 1450 | 1500 |
| Protein % | 28.5 | 27.5 | 23.6 | 20.0 | 15.7 |
| Fat % | 4.4 | 6.2 | 6.3 | 7.0 | 7.4 |
| Fiber % | 2.7 | 2.6 | 2.6 | 2.6 | 2.5 |
| Calcium % | 1.2 | 1.2 | 1.0 | 1.0 | 1.0 |
| Avail. P % | 0.6 | 0.6 | 0.5 | 0.5 | 0.5 |

SAMPLE COLLECTION AND HANDLING

Body weights were obtained at 1, 28, 56, and 119 days. At 4 and 8 weeks of age, each tom was placed on the scale within a cone box which had an opening on one side. The turkeys in each pen were herded into a corner with the use of a catching fence. Turkeys were returned to the pen immediately after recording the weight. During the weighing process at 17 weeks of age, each turkey was placed directly on the scale. Feed was weighed back on the day it was changed and feed efficiency was calculated on a per pen basis.

At 17 weeks of age, two of the leg banded turkeys were sacrificed from each pen. Both legs were removed at the proximal end of the tibia and frozen at -20$^0$C for latter measurement of bone strength. Two tubes of blood (one for

plasma and one for serum) were collected from all the leg banded turkeys at 7, 13, and 17 weeks of age, and sera and plasma were harvested and frozen, and three aliquots of sera were allocated for $1,25(OH)_2D_3$, testosterone and estradiol analysis, respectively, and plasma was used for Ca analysis.

## BONE STRENGTH TEST PROCEDURES

Twenty-four hours prior to mechanical testing, the tibia were thawed at room temperature and meat was removed.

Operation: Each tibia was subjected to four-point bending studies using a servohydraulic testing machine. The tibia were prepared for mechanical testing by passing two one-eighth inch pins through the bones. These served to support the bone throughout the test so that no movement could take place, except for rotation around the pin.

The neutral axis is an axis through the bone where there exists neither tension nor compression. For an accurate mechanical analysis of the forces in the bone, the neutral axis was visually approximated, and the two pins were drilled to pass through the axis. The lengths of the segments between the pins were measured using calipers. The tibia length (L) for each bone was taken to be the distance between the pins.

Bending loads were applied to each tibia until failure occurred. The hydraulic actuator displaced the central region of bone posteriorly with a haversian function, an amplitude of

one-half inch and a frequency of 1 Hertz. The displacement and maximum loads (P) were recorded on a Nicolet digital oscilloscope. A load versus deformation curve was then generated using an IBM personal computer.

The assumption was made that the tibia acted as a linear, elastic, uniform, hollow and elliptical beam with infinitesimal displacements in four-point bending.

According to the Elementary Strength of Materials Theory,

$$M \text{ (bending moment of the tibia)} = \frac{Pxa}{2}$$

where, $a = L/3$

and maximum tensile strength (sigma) can be calculated by:

$$\frac{Mxh}{2xI}$$

where h is vertical outside diameter of the tibia, I is the moment of inertia, and elastic modulus (E) can be measured by:

$$dP/dV\{a^2(3L-4a)/12I\}$$

where the dP/dV is the slope of the linear model.

## $1,25\ (OH)_2D_3$ TEST PROCEDURES

The Nichols Institute Diagnostics 1,25-Dihydroxyvitamin D Assay Kit was used for the test. Since circulating concentrations of $1,25\text{-}(OH)_2D_3$ were extremely low, a single serum extraction for $1,25\text{-}(OH)_2D_3$ was required before the

assay.

**EXTRACTION PROCEDURE:** The 12x75 mm borosilicate glass tubes were labeled for the extraction of each control and turkey sample. One ml of each control and turkey sample was pipetted into the appropriately labeled tubes. Fifty ul of recovery tracer was added to each tube and to two scintilation vials, capped, mixed, and set aside. Counts from these vials were used for calculating recovery. The control and turkey sample tubes were vortexed twice over the next 10 minutes. Then 1 ml acetonitrile was added to each tube. The tubes were vortexed vigorously for 30 seconds and then centrifuged at 1300-1500 x g for 15 minutes at 2-8 $^{0}$C. The supernatant was decanted into a 12 x 75 mm borosilicate glass tube containing 1 ml of 0.4 M potassium phosphate, PH 10.5 and vortexed, and then centrifuged at 1300-1500 x g for 10 minutes at 2-8 $^{0}$C. The sample was then decanted into a preconditioned $C_{18}$OH column and vacuum applied (5-7 mm Hg). Samples were allowed to run slowly (dripping) and completely through the columns. The columns were washed with 5 ml distilled water, 5 ml 70% methanol in distilled water, 5 ml 10% methylene chloride in hexane and 5 ml 1% isopropanol in hexane. Finally, 1,25-$(OH)_2D_3$ was eluted with 5 ml of 5% isopropanol in hexane. Elutes were dried with a nitrogen manifold in a hood using 5 psi of nitrogen. Dried elutes were reconstituted with 200 ul zero standard, vortexed and kept on ice. After 5 minutes, the dried elutes were rolled up to the level of elute before nitrogen

drying, vortexed again, waited another 5 minutes and then started assay within 30 minutes.

**ASSAY PROCEDURE:** Serum extracts were incubated with a sensitive 1,25-$(OH)_2D_3$ binding protein for one hour and then tritiated 1,25-$(OH)_2D_3$ was added. After one hour, dextran coated charcoal suspension was introduced to separate the bound and free tritiated 1,25-$(OH)_2D_3$. Subsequent to a 30 minutes incubation at 2-8 $^0C$, all tubes were centrifuged at 1500 x g for 20 minutes at 2-8 $^0C$. The supernatants were decanted to scintillation vials. Scintillation "cocktail" was added and the contents were mixed. The radioactivity of the tritiated protein-bound vitamin D was measured in a liquid scintillation counter. The concentration of 1,25-$(OH)_2D_3$ in serum was determined by comparison of counts from the sample vial with counts from the standard curve vials. Values were expressed in picograms per milliliter of serum after corrections for the sample concentration factor and the sample recovery from the column chromatography step.

## TOTAL TESTOSTERONE TEST (Coat-A-Count) PROCEDURES

The Coat-A-Count procedure is a solid-phase radioimmunoassay, based on testosterone-specific antibody immobilized to the wall of a polypropylene tube. $I^{125}$-labeled testosterone competes for a fixed time with testosterone in the turkey serum for antibody sites. The tube is then

decanted, to separate bound from free, and counted in a gamma counter. The amount of testosterone present in the turkey sample is determined from a standard curve.

Operations: First, four plain 12x75 polypropylene tubes were labeled for total counts and nonspecific binding in duplicate. Then, twelve total testosterone Antibody-Coated tubes were labeled for one maximum binding and five standards in duplicate. Additional antibody-coated tubes, also in duplicate, were labeled for controls and turkey samples. Then, 50 ul of the zero calibrator was pipetted into total count and nonspecific tubes, and 50 ul of each remaining calibrator, control and turkey sample were pipetted into the tube prepared; then 1.0 ml of $I_{125}$ total testosterone was added into each tube which was vortexed and incubated for 3 hours at $37^0$ C. Finally, all tubes were counted for 1 minute in a gamma counter after the contents of all tubes were decanted except the total count tubes and results were printed out.

ESTRADIOL TEST (DOUBLE ANTIBODY ESTRADIOL) PROCEDURES

The double antibody estradiol procedure is a sequential radioimmunoassay in which the turkey sample is preincubated with anti-estradiol antiserum. $I^{125}$-labeled estradiol then competes with estradiol in the turkey sample for antibody sites. After incubation for a fixed time, separation of bound-$I^{125}$ estradiol from the free is achieved by the PEG-accelerated

double-antibody method. Finally, the bound-antibody fraction is precipitated and counted. Turkey sample concentrations were read from a calibration curve.

Operations: Eighteen tubes were labeled in duplicate for 2 total counts, 2 nonspecific bindings, 2 maximum bindings, and 12 calibrators. Additional tubes, also in duplicate, were labeled for turkey sample and controls. Two hundred ul of the zero calibrator was pipetted into the nonspecific binding and maximum binding, and 200 ul of the remaining calibrators into correspondingly labeled tubes. Two hundred ul of each turkey serum sample and each control were then pipetted into the tubes prepared. Then, 100 ul of estradiol antiserum was added into all tubes except nonspecific binding and total count, and all tubes were vortexed and incubated for 2 hours at room temperature. Then, 100 ul of $I^{125}$ estradiol was added into all tubes. All tubes were vortexed and incubated for one hour at room temperature. Then 1.0 ml of cold precipitating solution was added into all tubes, and all tubes were vortexed and incubated for 10 minutes at room temperature. Thereafter, all tubes were centrifuged for 15 minutes at 3000 x g or 30 minutes at 1500 x g. The supernatant was decanted and the precipitate was retained for counting. Results were obtained after counting 1 minute in a gamma counter.

CALCIUM TEST PROCEDURES

Plasma Ca was measured by the 634 Ion Selective

Electrode $Ca^{++}$/PH Analyzer. The calcium ion selective electrode consists of a neutral carrier based calcium sensor. The sensor is in contact with the sample on one side, and with the electrode fill solution on the other side. Electrical connection is via a Ag/AgCl wire.

The test procedures include calibration of the analyzer first by using low (1.25mmol/L) and high (2.5mmol/L) standard Ca solutions. Then the probe was opened and dipped into the plasma sample tested. Then the probe was returned after plasma was sucked into the analyzer. Results were displayed within 45-70 seconds of returning the probe.

Feed efficiencies, body weights and average daily body weight gains were subjected to an one way analysis of variance. Non-leg weakness death, leg weakness death and total death were subjected to regular Chi Square test. Plasma calcium concentrations, serum $1,25\text{-}(OH)_2D_3$ and testosterone concentrations and bone strength were subjected to analysis of variance by the General Linear Model procedure, then Tukey's Studentized Range Test was used to determine which means were significantly different at the 0.01 and 0.05 levels of probability (Davies and Goldsmith, 1984).

# RESULTS AND DISCUSSIONS

Feed efficiencies (FE) of male turkeys at different ages raised under three different light treatments are summarized in Table 5. High intensity step-up (HISU), intermittent (INT) and control lighting treatments did not significantly influence FE of male turkeys during the three growth periods, as well as in the whole experiment from 0-17 wks of age although FE of male turkeys under HISU light (3.13±.2 kg of feed consumed/kg of body weight) and INT light (3.07±.05 kg of feed consumed/kg of body weight) were slightly better than those turkeys under control light (3.26±.06 kg of feed consumed/kg of body weight) between 9-17 wks of age.

FE of caponized turkeys at different ages raised under three different light treatments are shown in table 6. Caponization did not affect FE except between 9-17 wks of age when FE of capons under HISU light treatment (3.12 kg of feed consumed/kg of body weight) and INT light treatment (3.11 kg of feed consumed/kg of body weight) were slightly better than those capons under control light treatment (3.33 kg of feed consumed/kg of body weight).

**Table 5.** Feed efficiencies* of male turkeys at different ages raised under three different light treatments(kg of feed consumed/kg of BW)

| | Age | | | |
|---|---|---|---|---|
| Treatment | 0-4wks | 5-8wks | 9-17wks | 0-17wks |
| HISU | 1.35±.03[a**] | 1.76±.03[a] | 3.13±.20[a] | 2.61±.10[a] |
| INT | 1.34±.04[a] | 1.74±.03[a] | 3.07±.05[a] | 2.55±.02[a] |
| CNTL | 1.35±.02[a] | 1.77±.02[a] | 3.23±.06[a] | 2.66±.04[a] |

Wks stands for weeks.
HISU stands for high intensive step-up lighting treatment.
INT stands for intermittent lighting treatment.
CNTL stands for control lighting treatment.
*Feed efficiencies of male turkeys presented as mean ± SD.
** Values with same superscripts were not significantly different by Tukey's Studentized Range Test at $p<.05$ level.

**Table 6.** Feed efficiencies* of caponized turkeys at different ages raised under three different light treatments(kg of feed consumed/kg of BW)

| | Age | | | |
|---|---|---|---|---|
| Treatment | 0-4wks | 5-8wks | 9-17wks | 0-17wks |
| HISU | 1.42 | 1.83 | 3.12 | 2.58 |
| INT | 1.37 | 1.86 | 3.11 | 2.62 |
| CNTL | 1.34 | 1.83 | 3.33 | 2.71 |

Wks stands for weeks.
HISU stands for high intensive step-up lighting treatment.
INT stands for intermittent lighting treatment.
CNTL stands for control lighting treatment.
*There was only one pen of caponized turkeys for each treatment. Feed efficiencies of caponized turkeys were presented as actual numbers calculated and no statistics could be applied in this case.

Body weights (BW) of male turkeys raised under three different light treatments at different ages are summarized in Table 7. Significant differences ($P<0.05$) in BW were found among the three lighting treatments (TX). At 4 wks of age, the BW of male turkeys under INT light treatment was 1.12±.02 kg/bird which was significantly greater than those males under HISU light treatment (1.06±.03 kg/bird). However, no significant differences were found between HISU (1.06±.03 kg/bird) and CNTL (1.09±.03 kg/bird), as well as between INT (1.12±.02 kg/bird) and CNTL. At 17 wks of age, no significant differences were observed between HISU (13.58±.3 kg/bird) and CNTL (13.59±.3 kg/bird) although the INT bird had the heaviest BW (14.22 kg/bird).

BW of caponized turkeys at different ages are summarized in table 8. Like the BW of males, BW of capons under HISU (11.90 kg/bird) was similar to those capons under CNTL (12.84 kg/bird), and capons under INT light treatment had the heaviest BW (14.67 kg/bird) at 17 wks of age. But no big differences of BW were found among the light treatments at 4 and 8 wks of age in these capons.

Average daily body weight gains (ADBWG) of male turkeys raised under three different light treatments at different ages are summarized in Table 9. HISU, INT and CNTL light treatments did not affect ADBWG in all growth periods. The ranges of ADBWG for all three light treatments were very small: 41-43 g/bird/day between 0-4 wks of age, 61-63

**Table 7.** Body weights* of male turkeys at different ages raised under three light treatments (kg/bird)

| Treatment | Age | | |
|---|---|---|---|
| | 4wks | 8wks | 17wks |
| HISU | 1.06±.03$^{b**}$ | 3.39±.2$^{a}$ | 13.58±.3$^{b}$ |
| INT | 1.12±.02$^{a}$ | 3.51±.1$^{a}$ | 14.22±.4$^{a}$ |
| CNTL | 1.09±.03$^{ab}$ | 3.37±.1$^{a}$ | 13.59±.3$^{b}$ |

Wks stands for weeks.
HISU stands for high intensive step-up lighting treatment.
INT stands for intermittent lighting treatment.
CNTL stands for control lighting treatment.
*Body Weights of male turkeys were presented as mean ± SD.
**Values with different superscripts were significantly different by Tukey's Studentized Range Test at $p<.05$ level.

**Table 8.** Body weights* of caponized turkeys at different ages raised under three light treatments (kg/bird)

| Treatment | Age | | |
|---|---|---|---|
| | 4wks | 8wks | 17wks |
| HISU | 1.13 | 2.27 | 11.90 |
| INT | 1.04 | 3.01 | 14.67 |
| CNTL | 1.06 | 2.88 | 12.84 |

Wks stands for weeks.
HISU stands for high intensive step-up lighting treatment.
INT stands for intermittent lighting treatment.
CNTL stands for control lighting treatment.
*There was only one pen of caponized turkeys for each treatment. Body weights of caponized turkeys were presented as actual numbers calculated and no statistics could be applied in this case.

g/bird/day between 5-8 wks of age and 73-75 g/bird/day between 9-17 wks of age. During the whole study (0-17 wks), ADBWG of males under HISU and INT light treatment were 65±2 g/bird/day and 67±3 g/bird/day, respectively which were similar to that of those males under CNTL light treatment (64±1 g/bird/day). However, males grew much faster between 9-17 wks of age in which ADBWG of the males were almost twice as much as that between 0-4 wks of age in all three light treatments.

ADBWG of capons under three different light treatments are shown in table 10. Capons under HISU light treatment had a similar growth rate (55 g/bird/day) as those capons under CNTL light treatment (57 g/bird/day) between 0-17 wks of age, but capons under INT light grew fastest (64 g/bird/day). Caponized turkeys under all light treatments (except INT light treatment) grew slower than male turkeys after 4 wks of age. Therefore, these capons had lighter BW at 17 wks of age.

In the study, we found that HISU and INT light treatments did not significantly affect FE, BW, and ADBWG at 17 weeks of age compared with CNTL light treatment. In fact, the INT lighting treatment had significantly higher BW compared with CNTL light treatment. The results of our study were similar to those reported by Hester and Kohl (1989). It was indicated that FE and BW did not differ significantly between HISU and CNTL light treatments (Hester et al., 1983). Hulan et al. (1980) reported that turkeys, reared to 97 days of age in either INT light (4Lx2D), continuous light (23Lx1D),

**Table 9.** Average daily body weight gains* of male turkeys at different growth periods raised under three different light treatments (g/bird/day)

| | Age | | | |
|---|---|---|---|---|
| Treatment | 0-4wks | 5-8wks | 9-17wks | 0-17wks |
| HISU | $41\pm1^{a**}$ | $62\pm3^{a}$ | $74\pm4^{a}$ | $65\pm2^{a}$ |
| INT | $43\pm1^{a}$ | $63\pm4^{a}$ | $75\pm3^{a}$ | $67\pm3^{a}$ |
| CNTL | $42\pm2^{a}$ | $61\pm3^{a}$ | $73\pm2^{a}$ | $64\pm1^{a}$ |

Wks stands for weeks.
HISU stands for high intensive step-up lighting treatment.
INT stands for intermittent lighting treatment.
CNTL stands for control lighting treatment.
*Average daily body weight gains of male turkeys were presented as mean $\pm$ SD.
** Values with same superscripts were not significantly different by Tukey's Studentized Range Test at $p<.05$ level.

**Table 10.** Average daily body weight gains* of caponized turkeys at different growth periods raised under three different light treatments (g/bird/day)

| | Age | | | |
|---|---|---|---|---|
| Treatment | 0-4wks | 5-8wks | 9-17wks | 0-17wks |
| HISU | 35 | 48 | 68 | 55 |
| INT | 41 | 55 | 75 | 64 |
| CNTL | 41 | 51 | 65 | 57 |

Wks stands for weeks.
HISU stands for high intensive step-up lighting treatment.
INT stands for intermittent lighting treatment.
CNTL stands for control lighting treatment.
*There was only one pen of caponized turkeys for each treatment. Average daily body weight gains of caponized turkeys were presented as actual numbers calculated and no statistics could be applied in this case.

or total darkness, were shown to have no differences in FE, BW and grade A carcasses. The same patterns were reported by Gill and Leighton (1984) for male turkeys marketed at either 18 or 24 weeks of age. However, Buckland et al. (1974) examined the effect of INT lighting regimes on the performance of male turkeys and concluded that INT lighting programs were superior to the more conventional lighting regimes (14Lx10D or 16Lx8D). This was supported by the study of Siopes et al. (1986).

Non-leg weakness death, leg weakness death and total death of male turkeys are summarized in table 11, 12 and 13, respectively. HISU, INT and CNTL light treatments did not influence non-leg weakness death of the male turkeys in the whole study. However, more birds died due to leg weakness in the control light treatment (5.9±2.1 birds/pen) than did in the HISU light treatment (3.4±.5 birds/pen) and in the INT light treatment (3.4±1.4 birds/pen). Total death in the control was 9.1±3.3 birds/pen which was significantly reduced to 5.7±1 in HISU light treatment and to 7.4±3.6 birds/pen in INT light treatment ($P<0.05$).

Non-leg weakness death, leg weakness death and total death of caponized turkeys are indicated in table 14, 15 and 16. Caponization did not influence total death as measured in total numbers. However, improvement of the incidence of leg weakness death was observed in caponized birds under HISU light (1 birds/pen) and INT light (2 birds/pen) compared with capons under CNTL light (6 birds/pen).

**Table 11.** Non-leg weakness death of male turkeys* at different growth periods raised under three different light treatments (birds/pen)

| Treatment | Age | | | |
|---|---|---|---|---|
| | 0-4wks | 5-8wks | 9-17wks | 0-17wks |
| HISU | 1.3±1.4[a**] | 0.1±0.4[a] | 0.9±0.4[a] | 2.3±1.3[a] |
| INT | 2.0±1.0[a] | 0.6±0.8[a] | 1.4±2.1[a] | 4.0±3.3[a] |
| CNTL | 1.9±1.3[a] | 0.9±0.9[a] | 0.6±0.5[a] | 3.3±1.8[a] |

Wks stands for weeks.
HISU stands for high intensive step-up lighting treatment.
INT stands for intermittent lighting treatment.
CNTL stands for control lighting treatment.
*Non-leg weakness death of males presented as mean ± SD.
**Values with same superscript were not significantly different by Chi Square test at $p<.05$ level.

**Table 12.** Leg weakness death of male turkeys* at different growth periods raised under three different light treatments (birds/pen)

| Treatment | Age | | | |
|---|---|---|---|---|
| | 0-4wks | 5-8wks | 9-17wks | 0-17wks |
| HISU | 1.6±0.5[a**] | 0.4±0.5[a] | 1.4±0.5[a] | 3.4±0.5[a] |
| INT | 1.0±1.0[a] | 1.0±1.3[a] | 1.4±1.0[a] | 3.4±1.4[a] |
| CNTL | 3.1±2.0[b] | 0.6±0.5[a] | 2.1±0.4[a] | 5.9±2.1[b] |

Wks stands for weeks.
HISU stands for high intensive step-up lighting treatment.
INT stands for intermittent lighting treatment.
CNTL stands for control lighting treatment.
*Leg weakness death of males presented as mean ± SD.
**Values with different superscripts were significantly different by Chi Square test at $p<.05$ level.

**Table 13.** Total death of male turkeys* at different growth periods raised under three different light treatments (birds/pen)

| Treatment | Age | | | |
|---|---|---|---|---|
| | 0-4wks | 5-8wks | 9-17wks | 0-17wks |
| HISU | 2.9±1.3[a**] | 0.6±0.5[a] | 2.3±0.5[a] | 5.7±1.0[a] |
| INT | 3.0±0.8[a] | 1.6±1.4[a] | 2.9±2.0[a] | 7.4±3.6[ab] |
| CNTL | 5.0±2.8[b] | 1.4±1.1[a] | 2.7±0.5[a] | 9.1±3.3[b] |

Wks stands for weeks.
HISU stands for high intensive step-up lighting treatment.
INT stands for intermittent lighting treatment.
CNTL stands for control lighting treatment.
*Leg weakness death of males presented as mean ± SD.
**Values with different superscripts were significantly different by Chi Square test at $p<.05$ level.

**Table 14.** Non-leg weakness death of caponized turkeys* at different growth periods raised under three different light treatments (birds/pen)

| Treatment | Age | | | |
|---|---|---|---|---|
| | 0-4wks | 5-8wks | 9-17wks | 0-17wks |
| HISU | 1 | 3 | 0 | 4 |
| INT | 1 | 3 | 1 | 5 |
| CNTL | 0 | 2 | 0 | 2 |

Wks stands for weeks.
HISU stands for high intensive step-up lighting treatment.
INT stands for intermittent lighting treatment.
CNTL stands for control lighting treatment.
*There was only one pen of caponized turkeys for each treatment. Total death of caponized turkeys were presented as actual numbers calculated and no statistics could be applied in this case.

**Table 15.** Leg weakness death of caponized turkeys* at different growth periods raised under three different light treatments (birds/pen)

| Treatment | Age | | | |
|---|---|---|---|---|
| | 0-4wks | 5-8wks | 9-17wks | 0-17wks |
| HISU | 0 | 0 | 1 | 1 |
| INT | 0 | 0 | 2 | 2 |
| CNTL | 1 | 1 | 4 | 6 |

Wks stands for weeks.
HISU stands for high intensive step-up lighting treatment.
INT stands for intermittent lighting treatment.
CNTL stands for control lighting treatment.
*There was only one pen of caponized turkeys for each treatment. Total death of caponized turkeys were presented as actual numbers calculated and no statistics could be applied in this case.

**Table 16.** Total death of caponized turkeys* at different growth periods raised under three different light treatments (birds/pen)

| Treatment | Age | | | |
|---|---|---|---|---|
| | 0-4wks | 5-8wks | 9-17wks | 0-17wks |
| HISU | 1 | 3 | 1 | 5 |
| INT | 1 | 3 | 3 | 7 |
| CNTL | 1 | 3 | 4 | 8 |

Wks stands for weeks.
HISU stands for high intensive step-up lighting treatment.
INT stands for intermittent lighting treatment.
CNTL stands for control lighting treatment.
*There was only one pen of caponized turkeys for each treatment. Total death of caponized turkeys were presented as actual numbers calculated and no statistics could be applied in this case.

HISU and INT lighting treatments significantly reduced the incidence of leg weakness in this study in male turkeys. This was supported by the research of Hester et al. (1983) and Hester and Kohl (1989). Chernos (1988) also indicated that leg problems were improved 39% by HISU lighting treatment. The possible explanation was that HISU and INT lighting treatments stimulated secretion of testosterone which further stimulated synthesis of $1,25\text{-}(OH)_2D_3$. Then $1,25\text{-}(OH)_2D_3$ increased the absorption of calcium and phosphorus from the small intestine, the reabsorption from distal tubes of the kidney, and the mobilization of calcium and phosphorus from bones to provide enough calcium and phosphorus for bone mineralization and formation. An increase in exercise might also explain the reduction of leg abnormalities. Simons and Haye (1985), using radar equipment, showed that broilers reared in INT lighting were more active each hour during the light periods than broilers in continuous light. The increased feeding and drinking activities of turkeys in HISU and INT lighting treatments might have contributed to increased exercise, thus lowering the incidence of leg deformities.

The effect of lighting treatments on plasma calcium concentrations in both male and caponized turkeys can be seen in Table 17 and 18. The range of plasma concentrations was between 1.01 to 1.48 mmol/L. This finding is similar to previous reports by Frost and Roland (1991). No visible effects were observed during the first growth period. However,

significant differences ($P<0.05$) in plasma calcium concentrations were observed by 17 wks of age. The plasma calcium of male turkeys under INT treatment (1.48 mmol/L) was higher than that of the control (1.39 mmol/L). No significant differences of plasma calcium concentrations were observed in capons raised under the three light treatments at different ages.

It was believed that the higher plasma calcium concentrations observed in the birds under HISU and INT lighting treatment groups were the result of increased 1,25-$(OH)_2D_3$ concentrations. Frost and Roland (1990) reported that there were linear increases in serum calcium with increased levels of 1,25-$(OH)_2D_3$.

The influence of the lighting treatments on serum 1,25-$(OH)_2D_3$ concentrations in both male and caponized turkeys are shown in Table 19 and 20. The male turkeys under HISU lighting treatment had a significantly higher serum 1,25-$(OH)_2D_3$ ($41.5\pm.9$ pg/ml) than those males under the control light treatment ($37.4\pm.9$ pg/ml) at 17 wks of age. But lighting had no significant effect on male turkeys at 7 and 13 wks of age. Capons under INT ($105.0\pm18.8$ pg/ml) and CNTL ($103.0\pm15.9$ pg/ml) had significantly higher serum 1,25-$(OH)_2D_3$ concentrations than the capons under HISU light treatment ($65.6\pm7.1$ pg/ml) at 7 wks of age. However, lighting had no effect on serum 1,25-$(OH)_2D_3$ for capons at 13 and 17 wks of age.

**Table 17.** Plasma calcium concentrations of male turkeys* at different ages raised under three different light treatments (mmol/L)

| Treatment | Age | | |
|---|---|---|---|
| | 7wks | 13wks | 17wks |
| HISU | 1.02±.02[a**] | 1.12±.02[a] | 1.44±.02[ab] |
| INT | 1.02±.02[a] | 1.08±.02[ab] | 1.48±.02[a] |
| CNTL | 1.01±.02[a] | 1.03±.02[b] | 1.39±.02[b] |

Wks stands for weeks.
HISU stands for high intensive step-up lighting treatment.
INT stands for intermittent lighting treatment.
CNTL stands for control lighting treatment.
*Plasma calcium concentrations presented as mean ± SD.
** Values with different superscripts were significantly different by Tukey's Studentized Range Test at p<.05 level.

**Table 18.** Plasma calcium concentrations of caponized turkeys* at different ages raised under three different light treatments (mmol/L)

| Treatment | Age | | |
|---|---|---|---|
| | 7wks | 13wks | 17wks |
| HISU | 1.11±.08[a**] | 1.08±.08[a] | 1.45±.05[a] |
| INT | 1.03±.17[a] | 1.10±.11[a] | 1.49±.10[a] |
| CNTL | 1.00±.12[a] | 1.04±.25[a] | 1.48±.20[a] |

Wks stands for weeks.
HISU stands for high intensive step-up lighting treatment.
INT stands for intermittent lighting treatment.
CNTL stands for control lighting treatment.
*Plasma calcium concentrations presented as mean ± SD.
** Values with same superscripts were not significantly different by Tukey's Studentized Range Test at p<.05 level.

**Table 19.** Serum 1, 25-$(OH)_2D_3$ concentrations* of male turkeys at different ages raised under three different light treatments (pg/ml)

| Treatment | Age | | |
|---|---|---|---|
| | 7wks | 13wks | 17wks |
| HISU | $61.5\pm4.1^{a**}$ | $36.8\pm1.5^{a}$ | $41.5\pm0.9^{a}$ |
| INT | $59.7\pm4.1^{a}$ | $34.6\pm1.5^{a}$ | $38.0\pm0.9^{b}$ |
| LISD | $64.3\pm4.1^{a}$ | $39.5\pm1.8^{a}$ | $37.4\pm0.9^{b}$ |

Wks stands for weeks.
HISU stands for high intensive step-up lighting treatment.
INT stands for intermittent lighting treatment.
CNTL stands for control lighting treatment.
*Serum 1,25-$(OH)_2D_3$ concentrations presented as mean $\pm$ SD.
**Values with different superscripts were significantly different by Tukey's Studentized Range Test at $p<.05$ level.

**Table 20.** Serum 1, 25-$(OH)_2D_3$ concentrations* of caponized turkeys at different ages raised under three different light treatments (pg/ml)

| Treatment | Age | | |
|---|---|---|---|
| | 7wks | 13wks | 17wks |
| HISU | $65.6\pm 7.1^{b**}$ | $39.4\pm12.8^{a}$ | $44.2\pm5.9^{a}$ |
| INT | $105.0\pm18.8^{a}$ | $37.6\pm 2.2^{a}$ | $40.8\pm3.4^{a}$ |
| LISD | $103.0\pm15.9^{a}$ | $40.3\pm12.3^{a}$ | $43.4\pm8.7^{a}$ |

Wks stands for weeks.
HISU stands for high intensive step-up lighting treatment.
INT stands for intermittent lighting treatment.
CNTL stands for control lighting treatment.
*Serum 1,25-$(OH)_2D_3$ concentrations presented as mean $\pm$ SD.
**Values with different superscripts were significantly different by Tukey's Studentized Range Test at $p<.05$ level.

Serum 1,25-$(OH)_2D_3$ concentrations of our study were similar to those reported by Castillo et al. (1979). The high serum 1,25-$(OH)_2D_3$ concentrations could be due to the increased activity of 25-OH-$D_3$ 1-hydroxylase indirectly stimulated by the increased testosterone. Tanaka et al. (1976) and Castillo et al. (1977) reported that when estradiol was administered to mature male chickens, a stimulation of 25-OH-$D_3$ 1-hydroxylase activity was observed which was not seen if it was given to immature female or castrated male birds. When testosterone was given together with estradiol to castrated male birds, the 1-hydroxylase was also stimulated. The synergistic effect of estradiol and progesterone in stimulation of 1-hydroxylase was reported and testosterone, estradiol and progesterone given together gave the highest stimulation of this system (Tanaka et al., 1978). However, the mechanism of stimulation of 1-hydroxylase by the sex hormones in birds is not yet known nor is the reason for the dual requirement of estradiol and testosterone or progesterone. It is possible that the parathyroid glands mediate this stimulation, since parathyroid hormone is known to stimulate the 1-hydroxylase (Garabedian et al., 1972).

The serum 1,25-$(OH)_2D_3$ concentrations were higher in capons under INT and CNTL light treatments at 4 wks of age. The reason was not determined in this study.

Table 21 and 22 show the influence of lighting treatments and caponization on serum testosterone

concentrations in male and caponized turkeys. The serum testosterone could not be detected until the last growth period. The INT lighting treatment had higher serum testosterone concentrations (1.11 nmol/L) than did the control (0.55 nmol/L). Capons had zero serum testosterone concentration in all three light treatments.

The INT lighting treatment group had significantly ($p<0.05$) higher serum testosterone concentratons than did the control (LISD). Hester et al. (1983) also reported that the secondary sex characteristics of strutting and coloration of the head were more pronounced in toms of the HISU lighting throughout the latter part of the study. The increase of plasma testosterone concentrations and testes weights of toms in the HISU lighting program confirmed the visual observations that a faster rate of sexual maturity was being achieved. Absolute and relative testes weights were significantly greater for toms under INT lighting program than those receiving LISD lighting (Hester and Kohl, 1989). Gill and Leighton (1984) reported that INT lighting caused earlier sexual maturity in toms than the control lighting (12Lx12D). Broilers, 53 days of age, reared in the INT lighting program had larger testes than those under continuous light (Zakaria, 1987).

Table 23 illustrates the effect of lighting treatments on tibia breaking strength and maximum tensile strength. Lighting treatments did not influence these bone criteria.

**Table 21.** Serum testosterone concentrations* of male turkeys at 17 wks of age raised under three different light treatments (nmol/L)

| Treatment | Age |
|---|---|
| | 17wks |
| HISU | $0.87 \pm 0.1^{ab**}$ |
| INT | $1.11 \pm 0.1^{a}$ |
| CNTL | $0.55 \pm 0.1^{b}$ |

Wks stands for weeks.
HISU stands for high intensive step-up lighting treatment.
INT stands for intermittent lighting treatment.
CNTL stands for control lighting treatment.
*Serum testosterone concentrations presented as mean $\pm$ SD.
**Values with different superscripts were significantly different by Tukey's Studentized Range Test at $p<.05$ level.

**Table 22.** Serum testosterone concentrations* of caponized turkeys at 17 wks of age raised under three different light treatments (nmol/L)

| Treatment | Age |
|---|---|
| | 17wks |
| HISU | 0 |
| INT | 0 |
| CNTL | 0 |

Wks stands for weeks.
HISU stands for high intensive step-up lighting treatment.
INT stands for intermittent lighting treatment.
CNTL stands for control lighting treatment.
*There was only one pen of caponized turkeys for each treatment. Serum testosterone concentrations of caponized turkeys were presented as actural numbers calculated and no statistics could be applied in this case.

**Table 23.** Tibia breaking strength (Newtons) and maximum tensile strength* of turkeys raised under three different light treatments (Newtons/$mm^2$)

| Treatment | Tibia Breaking Strength | Maximum Tensile Strength |
|---|---|---|
| HISU | $1148\pm64^{a**}$ | $218\pm16^{a}$ |
| INT | $1111\pm64^{a}$ | $194\pm16^{a}$ |
| CNTL | $1015\pm64^{a}$ | $190\pm16^{a}$ |

Wks stands for weeks.
HISU stands for high intensive step-up lighting treatment.
INT stands for intermittent lighting treatment.
CNTL stands for control lighting treatment.
*Tibia breaking strength and maximum tensile strength presented as mean ± SD.
**Values with same superscript were not significantly different by Tukey's Studentized Range Test at $p<.05$ level.

Bone breaking strength relates to the size of bones and maximum tensile strength relates to the quality of bones, the content of calcium and phosphorus and the Ca/P ratio. Lighting treatments did not affect tibia breaking strength and maximum tensile strength in this study. Frost and Roland (1990) reported that tibia weight and tibia breaking strength were significantly increased by the stimulation of increased 1,25-$(OH)_2D_3$. Since there were only three samples used in each group for the study of tibia breaking and tensile strength, significant differences might be found if enough samples were used in each group.

In this trial, we were unable to detect serum estradiol by the technique that was utilized. This was due to the very low serum estradiol in the male turkeys at the ages sampled.

# SUMMARY

Commercially obtained day-old Nicholas toms were grown under HISU and INT lighting programs. HISU and INT light treatments did not affect the BW of turkeys when compared to the control. INT light treatment resulted in the heaviest BW.

Neither lighting treatment nor caponization affected tibia breaking strength and tibia maximum tensile strength in this study. More replicates should be used in future studies to obtain more sensitive data.

The incidence of leg abnormalities was significantly reduced by the HISU and INT lighting treatments without influencing FE and ADBWG when compared to the control lighting. In addition, HISU and INT lighting treatments significantly increased the serum testosterone, serum 1,25-$(OH)_2D_3$ and plasma calcium concentrations. It can be concluded that the decrease in leg weakness for the birds exposed to HISU and INT lighting was due to the increase of 1,25-$(OH)_2D_3$ synthesis stimulated by the increased serum testosterone.

Caponization significantly reduced the BW of turkeys in all lighting treatments except INT. The influence of caponization on leg weakness was inconclusive.

# APPENDIX

# APPENDIX

**Table 1.** Analysis of variance of feed efficiency for 0-4 wks male turkeys

| Source of variance | Degrees of freedom | Sum of squares | Mean square | F ratio |
|---|---|---|---|---|
| TX (A) | 2 | .0004 | .0002 | .25 |
| Rep (B) | 6 | .0040 | .0006 | .66 |
| A*B | 12 | .0100 | .0010 | |
| Total | 20 | | | |

TX stands for treatments.
REP stands for replications.

**Table 2.** Analysis of variance of feed efficiency for 5-8 wks male turkeys

| Source of variance | Degrees of freedom | Sum of squares | Mean square | F ratio |
|---|---|---|---|---|
| TX (A) | 2 | .003 | .0017 | .29 |
| Rep (B) | 6 | .001 | .0002 | .97 |
| A*B | 12 | .010 | .0010 | |
| Total | 20 | | | |

TX stands for treatments.
REP stands for replications.

**Table 5.** Analysis of variance of body weights for 5-8 wks male turkeys

| Source of variance | Degrees of freedom | Sum of squares | Mean square | F ratio |
|---|---|---|---|---|
| TX (A) | 2 | .089 | .044 | 4.03* |
| Rep (B) | 6 | .232 | .039 | 3.52* |
| A*B | 12 | .132 | .011 | |
| Total | 20 | | | |

TX stands for treatments.
REP stands for replications.
*P<.05.

**Table 6.** Analysis of variance of body weights for 9-17 wks male turkeys

| Source of variance | Degrees of freedom | Sum of squares | Mean square | F ratio |
|---|---|---|---|---|
| TX (A) | 2 | .357 | .178 | .84 |
| Rep (B) | 6 | .399 | .066 | .31 |
| A*B | 12 | 2.550 | .212 | |
| Total | 20 | | | |

TX stands for treatments.
REP stands for replications.

**Table 7.** Analysis of variance of average body weight gains per bird for 0-4 wks male turkeys

| Source of variance | Degrees of freedom | Sum of squares | Mean square | F ratio |
|---|---|---|---|---|
| TX (A) | 2 | 14.02 | 7.012 | 3.21 |
| Rep (B) | 6 | 7.39 | 1.232 | .56 |
| A*B | 12 | 26.21 | 2.184 | |
| Total | 20 | | | |

TX stands for treatments.
REP stands for replications.

**Table 8.** Analysis of variance of average body weight gains per bird for 5-8 wks male turkeys

| Source of variance | Degrees of freedom | Sum of squares | Mean square | F ratio |
|---|---|---|---|---|
| TX (A) | 2 | 20.05 | 10.03 | 1.37 |
| Rep (B) | 6 | 97.04 | 16.17 | 2.21 |
| A*B | 12 | 87.80 | 7.32 | |
| Total | 20 | | | |

TX stands for treatments.
REP stands for replications.

**Table 9.** Analysis of variance of average body weight gains per bird for 9-17 wks male turkeys

| Source of variance | Degrees of freedom | Sum of squares | Mean square | F ratio |
|---|---|---|---|---|
| TX (A) | 2 | 22.64 | 11.32 | .82 |
| Rep (B) | 6 | 27.23 | 4.54 | .33 |
| A*B | 12 | 166.20 | 13.85 | |
| Total | 20 | | | |

TX stands for treatments.
REP stands for replications.

**Table 10.** Analysis of variance of average body weight gains per bird for 0-17 wks male turkeys

| Source of variance | Degrees of freedom | Sum of squares | Mean square | F ratio |
|---|---|---|---|---|
| TX (A) | 2 | 21.05 | 10.53 | 1.72 |
| Rep (B) | 6 | 16.75 | 2.79 | .46 |
| A*B | 12 | 73.53 | 6.13 | |
| Total | 20 | | | |

TX stands for treatments.
REP stands for replications.

**Table 11.** Analysis of variance of plasma calcium concentrations for male turkeys

| Source of variance | Degrees of freedom | Sum of squares | Mean square | F ratio |
|---|---|---|---|---|
| TX (A) | 2 | .055 | .03 | 1.5 |
| Time (B) | 2 | 10.850 | 5.42 | 303.0** |
| Rep (C) | 34 | .610 | .02 | 1.0 |
| A*B | 4 | .239 | .06 | 3.3* |
| Total | 42 | 11.750 | | |

TX stands for treatments.
REP stands for replications.
*P<.05.
**P<.001.

**Table 12.** Analysis of variance of serum $1,25\text{-}(OH)_2D_3$ concentrations for male turkeys

| Source of variance | Degrees of freedom | Sum of squares | Mean square | F ratio |
|---|---|---|---|---|
| TX (A) | 2 | 494 | 247 | 1.02 |
| Time (B) | 2 | 38938 | 19469 | 80.10** |
| Rep (C) | 34 | 6098 | 179 | 0.74 |
| A*B | 4 | 554 | 139 | 0.57 |
| Total | 42 | 46505 | | |

TX stands for treatments.
REP stands for replications.
**P<.001.

**Table 13.** Analysis of variance of serum testosterone oncentrations for male turkeys

| Source of variance | Degrees of freedom | Sum of squares | Mean square | F ratio |
|---|---|---|---|---|
| TX (A) | 2 | 5.6 | 2.8 | 3.9* |
| Time (B) | 0 | 0 | 0 | 0 |
| Rep (C) | 34 | 19.4 | 0.6 | 0.8 |
| A*B | 0 | 0 | 0 | 0 |
| Total | 42 | 25.0 | | |

TX stands for treatments.
REP stands for replications.
*P<.05.

**Table 14.** Analysis of variance of turkey bone strength

| Source of variance | Degrees of freedom | sum of squares | Mean square | F ratio |
|---|---|---|---|---|
| Total | 17 | 439869 | | |
| Subclass | 3 | 91927 | 30642 | 1.23 |
| Treatment | 2 | 56282 | 28141 | 1.13 |
| Sex | 1 | 35644 | 35644 | 1.43 |
| Error | 14 | 347941 | 24853 | |

**Table 15.** Analysis of variance of turkey maximum tensile strength

| Source of variance | Degrees of freedom | Sum of square | Mean square | F ratio |
|---|---|---|---|---|
| Total | 17 | 26308 | | |
| Subclass | 3 | 4020 | 1340 | .84 |
| Treatment | 2 | 2852 | 1426 | .90 |
| Sex | 1 | 1168 | 1168 | .73 |
| Error | 14 | 22287 | 1592 | |

# REFERENCES

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Abbott, W. W., J. R. Crouch, and Atkinson, 1969. The incidence of foot pad dermatitis in young turkeys fed high levels of soybean meal. Poultry Sci. 48:2186-2188.

Adams, M. H., A. L. Waldroup, and P. W. Waldroup, 1993. Effect of time of protein restriction and amino acid level of realimentation diets on performance and leg strength of different strains of large white turkeys. Poultry Sci. 72:151.

Adams, R. L., and W. J. Stadelman, 1978. Effect of delay of growth on leg weakness of turkeys. Proc. 16th World Poult. Cong., Brazil. Vol. IV:559-565.

American Association of Avian Pathologists, 1987. 1986 summary of commercial poultry disease reports. Avian Dis. 31:926-982.

Akpobome, G. O., and R. C. Fanguy, 1989. The effect of high density on the performance of commercial broilers in cages. Poultry Sci. 68, Supplement 1:2.

Antillon, A., M. L. Scott, L. Krook, and R. H. Wasserman, 1988. Metabolic response of laying hens to different dietary levels of calcium, phosphorus and vitamin $D_3$. Cornell Vet. 67:413-414.

Asmundoson, V. S., 1942. An inherited micromelia in the domestic fowl. J. Hered. 33:328-330.

Asmundoson, V. S., 1944. Inherited shortening of the long bones in the turkey. J. Hered. 35:295-299.

Austic, R. E., D. J. Backer, and R. K. Cole, 1977. Susceptibility of a dwarf strain of chickens to rickets. Poultry Sci. 56:285-291.

Bagley, R., 1971. Osteomyelitis-importance in the turkey leg weakness syndrome. The Proc. of the Symposium on Leg Weakness in Turkeys. p 39.

Bell, D. J., and W. G. Siller, 1962. Cage layer fatigue in brown leghorns. Res. Vet. Sci. 3:219-229.

Brenes, J., 1971. Influence of calcium, phosphorus and vitamin $D_3$ on performance and bone metabolism of turkeys. Poultry Sci. 50:1557.(Abstr.)

Buckland, R. B., 1975. The effect of intermittent lighting programs to the production of market chickens and turkeys. World's Poult. Sci. J. 31:262-270.

Buckland, R. B., A. T. Hill, and D. E. Bernon, 1973. Effects of four lighting regimes on the performance of broilers and roasters. Can. J. Anim. Sci. 53:21-24.

Buckland, R. B., D. E. Bernon and A. Goldrosen, 1976. Effect of four lifting regimes on broiler performance, leg abnormalities and plasma corticoid levels. Poultry Sci. 55:1072-1076.

Buckland, R. B., N. J. Supeene and D.E. Bernon, 1974. Comparison of intermittent lighting programs for producing both broilers and heavy turkeys. Proc. Abst. XV World's Poult. Congr., New Orleans, LA. pp 425-427.

Buffington, D. E., K. A. Jordan, L. L. Boyd, and W. A. Junnila, 1973. Mathematical models of growth data of male and female Wrolstad white turkeys. Poultry Sci. 52:1694-1700.

Buffington, D. E., S. H. Cleven, and K. A. Jordan, 1975. The incidences of leg and foot abnormalities in Wrolstad White Turkeys. Poultry Sci. 54:457-461.

Candlish, J. R., 1981. The formation of mineral and organic matrix of fowl cortical and medullary bone during shell calcification. Br. Poult. Sci. 12:119-127.

Cantor, A. H., M. A. Musser, W. L. Bacon, and A. B. Hellewell, 1980. The use of bone mineral mass as an indicator of vitamin D status in turkeys. Poultry Sci. 59:563-568.

Caskey, C. D., W. D. Gallup, and L. C. Norris, 1939. The need for manganese in bone development of the chickens. Avian Dis. 8:48-55.

Castillo, L., Y., Tanaka, and H. F. Deluca, 1977. The stimulation of 25-hydroxyvitamin $D_3$-1-alpha-hydroxylase by estrogen. Arch Biochem Biophys. 179:211.

Castillo, L., Y.,Tanaka, M. J. Wineland, J. O. Jowsey, and H. F. Deluca, 1979. Production of 1,25-$(OH)_2D_3$ and formation of medullary bone in the egg-laying hen. Endocrinology. 104:1598-1601.

Charles, O. W., and J. Fortune, 1977. The influence of diet and litter management on foot pad lesions in turkey poults. Poultry Sci. 56:1348. (Abstr.).

Chernos, R., 1988. Broilers on six hours of light. Canada Poultryman. August:6-7.

Classen, H. L., and C. Riddell, 1989. Photoperiodic effects on performance and leg abnormalities in broiler chickens. Poultry Sci. 68:873-879.

Clayton, G. A., C. Nixey, and G. Monaghan, 1978. Meat yield in turkeys. Br. Poult. Sci. 19:755-763.

Clements, M. R., L. Johnson, and D. R. Fraser, 1987. Nature. 325:62-65.

Collins, E. D., and A. W. Norman, 1991. Vitamin D. Handbook of Vitamins. pp 69-85.

Cook, M. E., P. H. Patterson, and M. L. Sunde, 1984. Leg deformities: inability to increase severity by increasing body weight of chicks and poults. Poultry Sci. 63:620-627.

Cook, M. E., W. T. Springer, and J. A. Herbert, 1984. Enhanced incidence of leg abnormalities in reovirus WVU2937 infected chickens fed various dietary levels of selected vitamins and minerals. Avian Dis. 28:548-561.

Cope, F. O., M. Stuart, and P. E. Stake, 1979. Reduction in the incidence and severity of curled toes and perosis-like leg abnormalities in caged-reared broilers by high dietary levels of pyridoxine or choline. Poultry Sci. 58:1046. (Abstr.)

Couch, J. R., Cage Layer Fatigue, 1955. Feed Age. 5:55-57.

Daniel, L. J., F. A. Farmer, and L. C. Norris, 1946. Folic acid and perosis. J. Biol. Chem. 163:349-350.

Davies, O. L. and P. L. Goldsmith, 1984. Statistical Methods in Research and Production. pp 152-153.

Deluca, H. F., 1980. Vitamin D hormonal system. Pharmacological Modulation of Steroid Action, Raven Press, New York. pp 11-31.

Deluca, H. F., and H. K. Schnoes, 1983. Ann. Rev. Biochem. 52:411-439.

Dobson, D. C., 1970. Biotin requirement of turkey poults. Poultry Sci. 49:546-553.

Edwards, H. M., Jr., 1989. The effect of dietary cholecalciferol, 25-hydroxycholecalciferol, and 1,25-dihydroxycholecalciferol on the development of tibial dyschondroplasia in broiler chickens in the absence and presence of disulthiram. J. Nutr. 119:647.

Etches, R. J., 1987. Calcium logistics in the laying hens. J. Nutr. 117:619-628.

Ferket, P. R., and J. L. Sell, 1989. Effect of severity of early protein restriction on large turkey toms. I. Performance characteristics and leg weakness. Poultry Sci. 68:676-686.

Francis, D. W., 1957.Strain differences in the incidence of cage layer fatigue. Poultry Sci. 36:181-183.

Frost, T. J. and D. A. Roland, Sr., 1990. Influence of vitamin $D_3$, and 1,25-$(OH)_2D_3$ on eggshell quality, tibia strength, and various production parameters in commercial laying hens. Poultry Sci. 69:2008-2016.

Frost, T. J. and D. A. Roland, Sr., 1991. The influence of various calcium and phosphorus levels on tibia strength and eggshell quality of pullets during peak production. Poultry Sci. 70:963-969.

Garabedian, M., M. F. Holick, H. F. Deluca, and I. T. Boyle, 1972. Control of 25-hydroxycholecalciferal metabolism by the parathyroid glands. Proc Natl Acad Sci. 69:1673.

Gill, D. J., and A. T. Leighton, Jr., 1984. Effect of light environment and population density on growth performance of male turkeys. Poultry Sci. 63:1314-1321.

Glass, S. E., 1971. Dianostic laboratory observations on the incidence and significance of various leg weakness syndromes. The Proc. of the Symposium on the Leg Weakness in Turkeys. pp 58-62.

Gries, C. L., and M. L. Scott, 1972. The pathology of pyridoxine deficiency in chicks. J. of Nutr. 102:1259-1267.

Harms, R. H., and C. C. Simpson, 1977. Influence of wet litter and supplemental biotin on foot pad dermatitis in turkey poults. Poultry Sci. 56:2009-2012.

Harshaw, H. M., and R. R. Rector, 1940. The composition of turkeys as affected by age and sex. Poultry Sci. 19:404-411.

Haussler, M. R., and T. A. McCain, 1977. N. Engl. J. Med. 297:974-983.

Haye, U., and P. C. M., Simons, 1978. Twisted legs in broilers. Br. Poult. Sci. 19:549-557.

Henry, H. L., and A. W. Norman, 1984. Ann. Rev. Nutr. 4:493-520.

Hester, P. Y., A. L. Sutton, and R. G. Elkin, 1986. The effect of light intensity, light source and litter management on the incidence of leg abnormalities and performance of turkey toms. Poultry Sci. 66:666-675.

Hester, P. Y., A. L. Sutton, R. G. Elkin, and P. M. Klingensmith, 1985. The effect of lighting, dietary amino acids and litter on the incidence of leg abnormalities and performance of turkey toms. Poultry Sci. 64:2062-2075.

Hester, P. Y., and H. Kohl, 1989. Effect of intermittent lighting and time of hatch on large broad breasted turkeys. Poultry Sci. 68:528-538.

Hester, P. Y., R. G. Elkin, and P. M. Klingensmith, 1983. Effects of high intensity step-up and low intensity step-down lighting programs on the incidence of leg abnormalities in turkeys. Poultry Sci. 62:887-896.

Hilley, H. D., 1982. Skeletal abnormalities in the pigs. Vet. Clin. North. Am. 4:225.

Hogan, A. G., L. R. Richardson, H. Patrick, and H. L. Kemster, 1941. Perosis due to a vitamin deficiency. J. Nutr. 21:327-340.

Hulan, H. W., and F. G. Proudfoot, 1987. Effects of light source, ambient temperature and diet energy source on the general performance and incidence of leg abnormalities of roaster chickens. Poultry Sci. 66:645-651.

Hulan, H. W., F. G. Proudfoot, and K. B. McRae, 1980. The nutritional value of tower and candle rapeseed meals for turkey broilers housed under different lighting conditions. Poultry Sci. 59:100-109.

Hurwitz, S., 1965. Calcium turnover in different bone segments of laying hens. Am. J. Physiol. 208:203-207.

Hurwitz, S., and A. Bar, 1969. Intestinal calcium absorption in the laying fowl and its importance in calcium homeostasis. Amer. J. Clin. Nutr. 22(4):391-395.

Jensen, L. S., and R. Martinson, 1969. Requirements of turkey poults for biotin and effect of deficiency on incidence of leg weakness in developing turkeys. Poultry Sci. 48:222-230.

Johnson, A. L., and V. T. Rendano, 1984. Effects of castration, with and without testosterone replacement, on leg bone integrity in the domestic fowl. Am. J. Vet. Res. 45:319-325.

Johnson, A. L., 1987. Reproduction in the Male. Avian Physiology. pp 432-451.

Jubb, K. V. F., and P. C. Kennedy, 1970. Pathology of domestic animals. Vol. I. 2nd edit. Academic press, NY, USA.

Jukes, T. H., 1940. Prevention of perosis by choline. J. Biol. Chem. 134:789-790.

Julian, R. J., 1990. Musculo-skeletal disease in meat-type poultry. Avian Skeletal Disease Symposium, pp 5-8.

Ketelaars, E. H., M. Verbrugge, W. Van derHel, J. M. Van de Linden, and W. M. A. Veerstegen, 1986. Effect of intermittent lighting on performance and energy metabolism of broilers. Poultry Sci. 65:2208-2213.

Kramer, S. L., and P. E. Waibel, 1978. The vitamin D requirement of turkey breeder hens in cages. pp119-123 in: Minnesota Turkey Res. Bull.

Laing, P. W., 1976. Lameness and leg weakness in rapidly growing turkeys associated with hip lesions. Vet. Res. 99:391-392.

Leach, R. M., Jr., and E. G. Buss, 1977. The effect of inherited chondrodystrophy on the hexosamine content of cartilage from turkey embryos. Poultry Sci. 56:1043-1045.

Leach, R. M., Jr., and M. C. Nesheim, 1965. Nutritional, genetic, and morphological studies of an abnormal cartilage formation in young chicks. J. Nutr. 86:236-244.

Marsden, S. J., 1940. Weights and measurements of parts and organs of turkeys. Poultry Sci. 19:23-28.

Martland, M. F., 1985. Ulcerative dermatitis in broiler chickens: the effect of wet litter. Avian Pathol. 14:353-364.

McCarthy, J. T., S. S. Barham, and R. Kumar, 1984. J. Steroid Biochem. 21:253-265.

McGinnis, J., and J. S. Carver, 1946. The effect of riboflavin and biotin on dermatitis and perosis in turkey poults. Poultry Sci. 25:407-408.

Miller, B. F., 1968. Comparative yield of different size turkey carcasses. Poultry Sci. 47:1570-1574.

Miller, S. C., 1981. Osteoclast cell-surface specializations and nuclear kinetics during egg-laying in Japanese quail. Am. J. Anat. 162:35-43.

Mueller, W. J., R. Schroer, and H. Schroer, 1977. Calcium metabolism and skeletal dynamics of laying pullets. J. Nutr. 84:20-26.

National Research Council, 1984. Nutrient requirements of turkeys. pp 16-18.

Nestor, K. E., 1984. Genetics of growth and reproduction in the turkey, 9 long-term selections for increased 16 week body weight. Poultry Sci. 63:2114-2122.

Nestor, K. E. and D. A. Emmerson, 1990. Role of genetics in expression and prevention of leg weakness. Avian Skeletal Disease Symposium, pp 14-21.

Nestor, K. E., W. L. Bacon, Y. M. Saif, and P. A. Renner, 1985. The influence of genetic increases in shank width on body weight, walking ability, and reproduction of turkeys. Poultry Sci. 64:2248-2255.

Nestor, K. E., W. L. Bacon, P. D. Moorhead, Y. M. Sait, G. B. Havenstein, and P. A. Renner, 1987. Comparison of bone and muscle growth in turkey lines selected for increased body weight and increased shank width. Poultry Sci. 66:1421-1428.

Nestor, K. E., Y. M. Saif, and P.A. Remer, 1982. Genetics of leg weakness in turkeys. Ohio Turkey Days. p 40.

Norman, A. W., J. Roth, and L. Orci, 1982. Endocrinol. Rev. 3:331-336.

Norris, L. C., 1971. The turkey leg weakness problem. The Proc. of the Symposium on Leg Weakness in Turkeys. pp 58-62.

Olsson, N., 1950. Investigations on the vitamin D requirements of chicks, poults, ducklings and goslings. World's Poultry Sci. J. 6:230.

Olson, N. O., 1971. Experimental mycoplasma synoviae infection in turkeys. The Proc. of the Symposium on Leg Weakness in Turkeys. pp 28-29.

Patrick, H, R. V. Boucher, R. A. Dutcher, and H.. C. Knandel, 1942. The nutritional significance of biotin in chick and poult nutrition. Poultry Sci. 21:467.

Plavnik, I., and S. Hurwitz, 1985. The performance of broiler chicks during and following a severe feed restriction at an early age. Poultry Sci. 64:348-355.

Pomeroy, B. S., S. H. Kleven, and A. Peterson, 1971. Mycoplasma infection in turkeys. The Proc. of the Symposium on Leg Weakness in Turkeys. pp 26-27.

Poss, P. E. and J. B. Johnson, 1971. Osteomyelitis in turkeys caused by coliforms. The Proceedings of the Symposium on Leg Weakness in Turkeys. p 40.

Poulos, P. W., S. Reiland, K. Elwinger, and S.-E., 1978. Skeletal lesions in the broiler, with special reference to dyschondroplasia. Acta Radiologica Supplement 358:229-275.

Reiland, S., 1978. Pathology of so-called leg weakness in the pig. Acta. Radiol. Suppl. 358:153.

Rejino, S., and B. Stromberg,1978. Osteochondrosis in the horse. Acta. Radiol. Suppl. 358:153.

Riddell, C., 1975. Studies on the pathogenesis of tibial dyschondroplasia in chickens. II. Growth rate of long bones. Avian Diseases. 19:490-496.

Riddell, C., 1980. A survey of skeletal disorders in five turkey flocks in Saskatchewan. Can. J. Comp. Med. 44:275-279.

Riddell, C., 1983. Pathology of the skeleton and tendons of broiler chickens reared to roaster weights. I. Crippled chickens. Avian Diseases 27:950-962.

Riddell, C., 1990. Effect of management on skeletal problems in poultry. Avian Skeletal Disease Symposium. pp 60-67.

Riddell, C., C. F. Helmboldt, E. P. Singsen, and L. D. Mattersen, 1968. Bone pathology of birds affected with cage layer fatigue. Avian Dis. 12:285-296.

Riddell, C., 1989. Cage layer fatigue-A rev. Proc. 38th Western Poultry Conf., Tempe, Arizona. pp 87-88.

Rimoin, D. L., D. W. Hallister, R. S. Lackman, W. H. McAllister, R. E. Rosenthal, and G. F. Hughes, 1974. Histologic studies in the chondrodystrophies. Birth Defects 10:274

Rizk, S. W., P. E. Stake, and R. W. Simmons, 1980. Curled toes and perosis-like abnormalities in cage-reared broilers. Poultry Sci. 59:308-315.

Rodenhoff, G.,and K. Dammrich, 1973. Untersuchungen zur beeinflussung der rohrenknochenstruktur durch verschiedene haltungssysteme bei masthahnchen. Berliner und muencher Tieraerztliche Wochenschrift 86:230-233, 241-244.

Sange, V. L., R. R. Dahlgren, M. S. Cover, and R. F. Langham, 1974. Skeletal disease and locomotor problems in turkeys. Avian Diseases. 18:379-393.

Sauver, B., 1984. Dietary factors as causes of leg abnormalities in poultry-A Rev. World' Poult. Sci. J. 63:620-627.

Schwartz, L. D., 1988. Poultry Health Handbook. pp 126-127.

Scott, M. L., 1987. Vitamin D. Nutrition of the Turkey. pp 110-114.

Scott, M. L., 1987. Nutrition of the Turkey. p 109.

Scott, M. L., 1987. Nutrition of the Turkey. p 111.

Scott, M. L., M. C. Necheim, and R. J. Young, 1976. Calcium and phosphorus. Nutrition of the Chickens. pp 84-300.

Scott, M. L., M. C. Necheim, and R. J. Young, 1982. Nutrition of the chicken. M. L. Scott and Associates, Ithaca, NY.

Siegel, H. S., L. N. Drury, and W. C. Patterson, 1973. Bone characteristics and growth of broilers housed in plastic coops or on litter in moderate and high temperature. 4th European Poultry Conference, London. pp 159-164.

Siller, W. G., 1970. Tibial dyschondroplasia in the fowl. J. Pathol. Bacteriol. 101:39-46.

Simkiss, R., 1967. Calcium in reproductive physiology. A comparative study of vertebrate. Chapman and Hall Ltd., London.

Simons, P. C. M., 1982. Effect of lighting regimes on twisted legs, feed conversion and growth of broiler chickens. Poultry Sci. 61:1546.(Abstr.)

Simons, P. C. M., 1986. The incidence of leg problems as influenced by management. Proc. 7th European Poultry Science Symposium, Paris. pp 289-299.

Simons, P. C. M., and U. Haye, 1985. Intermittent lighting has a positive effect on twisted leg. Poultry Sci. March:34-37.

Soares, J. H., Jr., 1984. Calcium metabolism and its control-A rev.. Poultry Sci. 63:2075-2083.

Somes, R. G., Jr., 1969. Genetic perosis in the domestic fowl> J. Hered. 60:163-166.

Siopes, T. D., C. R. Parkhurst, and G. R. Baughman, 1986. Intermittent light and growth performance of male turkeys from 2 to 22 weeks of age. Poultry Sci. 65:2221-2225.

Stevens, I., R. Blair, R. E. Salmon, and J. P. Stevens, 1984. Effects of varying levels of dietary vitamin D on turkey hen egg production, fertility and hatchability, embryo mortality and incidence of embryo beak malformation. Poultry Sci. 63:760-764.

Tanaka, Y., L. Castillo, and H. F., Deluca, 1976. Control of renal vitamin C hydroxylase in birds by sex hormones. Proc Natl Acad Sci. 73:2701.

Tanaka, Y., L. Castillo, M. J. Wineland, and H. F. Deluca. 1978. Synergistic effect of estradiol, testosterone and progesterone in the stimulation of chick renal 25-hydroxyvitamin $D_3$-1-alpha-hydroxylase. Endocrinology. 103:2035.

Taylor, T. G., and A. Williams, and J. Kirkley, 1965. Cyclic changed in the activities of plasma acid and alkaline phosphatase during egg shell calcification in the domestic fowl. Can. J. Biochem. 43:451-457.

Underwood, J. L., and H. F. Deluca, 1984. Am. J. Physiol. 246:E493-498.

Veltmann, J. R. Jr., and L. S. Jensen, 1979. Variation in incidence of tibial dyschondroplasia in broiler chicks associated with environmental conditions. Poultry Sci. 58:1118.

Veltmann, J. R., Jr., G. N. Rowland, and S. S. Linton, 1985. Tibial dyschondroplasia in single-comb white leghorn chicks fed tetraethylthiuram disulfide. Avian Dis. 29:1269-1272.

Wideman, R. F., 1987. Renal regulation of avian calcium and phosphorus metabolism. J. Nutr. 117:808-815.

Wilgus, H. S., 1931. The quantitative requirement of the growing chick for calcium and phosphorus. Poultry Sci. 10:107.

Wilson, J. C., W. D. Weaver,W. L. Beane, and J. A. Cherry, 1984. Effect of light and feeding space on leg abnormalities in broilers. Poultry Sci. 63:565-567.

Wise, D. R., A. R. Jennings, and D. E. Bostock, 1973. Perosis in turkeys. Res. Vet. Sci. 14:167-172.

Wise, D. R., 1975. Skeletal abnormalities in table poultry-A review. Avian Pathol. 4:1-10.

Wise, D. R., and H. Nott, 1975. Studies on tibial dyschondroplasia in ducks. Res. Vet. Sci. 18:193-197.

Wu, W., 1990. Etiology and physiology of chemical-induced tibial dyschondroplasia in broiler chickens. Ph.D. Dissertation, University of Wisconsin, Madison, WI 53706.

Wu, W, M. E. Cook, and E. B. Smalley, 1991. Decreased immune response and increased incidence of tibial dyschondroplasia caused by Fusaria grown on sterile corn. Poultry Sci. 70:293-301.

Yang, H. S., P. E. Waibel and J. Brenes, 1973. Evaluation of vitamin $D_3$ supplements by biological assay using the turkey. J. Nutrition 103:1187-1194.

Young, H. S., P. E. Waibel, and J. Brenes, 1973. Evaluation of vitamin $D_3$ supplements by biological assay using the turkey. J. Nutrition. 103:1187-1194.

Young, R. J., H. M. Edwards,Jr., and M. B. Gillis, 1958. Studies on zinc in poultry nutrition 2. Zinc requirement and deficiency symptoms of chicks. Poultry Sci. 37:1100-1107.

Zakaria, A. H., 1987. Sex differences in the effect of intermittent compared with continuous light on growth rate, testes and body weight of broilers reared under commercial condition. Br. Poult. Sci. 28:343-346.