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Technical Bulletin

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Leg Weakness/Disorders in Poultry

Modern poultry farming, that has transformed from a mere backyard rearing activity to a vibrant and dynamic industry over the past few decades, has resulted in the tremendous increase in the growth rate of poultry. The demand for increased growth rate and production due to the economic benefits of higher body weight and increased egg production respectively has led to the differential growth of body parts. In particular the accelerated growth of muscle. However this growth isn't accompanied by the skeletal development thus imparting more strain on the skeletal integrity

The leg bones are the fastest growing bones in the body. Rapid growth rate accompanied by adequate mineral and vitamin nutrition, proper management, an optimum lighting and temperature program, a disease free environment prevents weakness/disorders in birds. The absence of any of the above factors combined with the intrinsic weight bearing characteristics of the individual birds give rise to the different degrees of leg disorders.

Leg weakness can be classified as infectious, developmental/metabolic and degenerative disorders. Young rapidly growing broiler chicks are commonly affected with disorders affecting the bone growth while adolescent and adult breeding stock of meat-type birds suffer from degenerative joint disorders. The commercial layers and layer breeders are more prone to conditions that lead to bone fragility. Leg weakness leads to high incidence of morbidity than mortality. In general, they have an incidence varying from 2 to 15%, which inflict a considerable monetary loss to the producer in terms of culling on the farm and/or condemnations or downgrading at processing (Knowles and Wilkins, 1998). Leg weakness also represents animal welfare concern (Webster, 1995; Weeks and Kestin, 1997) in which the disabled bird experiences pain, does not reach feed and water and dies due to inanition. From the aesthetic point of view, fragile and porous bones lead to the presence of bone fragments in deboned meat products along with discolouration of meat adjacent to the bone due to leaching of blood, making the product less appealing to the consumer (Rath *et al*., 2000).

BONE STRUCTURE

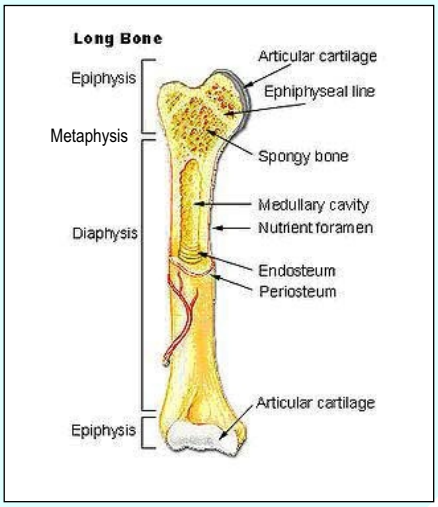
The major mineral reserve in the body of a chicken is the bone which is a collagenous connective tissue. The bone becomes rigid under conditions of proper mineralization and vascularization. The skeleton dictates the shape and stature of the bird and provides support to soft tissues and muscle. The main mineral component of the bone is calcium phosphate (ratio of 2:1 for Ca and P). A normal bone consists of 99% calcium, 88% phosphate, 80% bicarbonate, 50% magnesium and 35% of total mineral body reserves. There are three types of bone in chicken, cortical or structural bone found in young as well as adult birds; cancellous bone, a form of structural bone that has more metabolic function and undergoes continuous remodeling as found in the skull, vertebral and metaphyseal ends of long bones (Seifert and Watkins, 1997) and medullary or spongy bone found in mature female birds which act as a reservoir of labile calcium during egg formation.

A bone consists of three separate regions namely epiphysis, metaphysis and diaphysis (Fig. 1). Metaphysis is the area of most active bone growth and controls the longitudinal growth and thickness of the bone. The entire bone is surrounded by periosteum, rich in capillaries that aids in the nourishment of the bone. Bone calcification is aided by the osteoblasts and excessive thickening is prevented by osteoclasts which reabsorb the worn out cells. Hence, normal development depends on the continual deposition and resorption of the bone. Bone formation during fetal stage occurs by two processes namely intramembranous and endochondral ossification. The intramembranous ossification occurs during formation of flat bones in the skull and consists of development of ossification center, calcification, trabeculae formation and development of periosteum. Most of the bone growth particularly that of long bones occurs by endochondral ossification and the steps involved are development of the cartilage model, growth of cartilage model, development of the primary and secondary ossification center and formation of articular cartilage and epiphyseal plate.

A bone is approximately 70% inorganic (minerals), 20% organic (collagen, proteoglycan, lipids and non-collagenous proteins) and 10% water.

The inorganic (calcium phosphate-hydroxyapatite) and organic (Type-1 collagen) matrix imparts compression and tensile strength to the bone respectively.

Fig. 1: Structure of bone

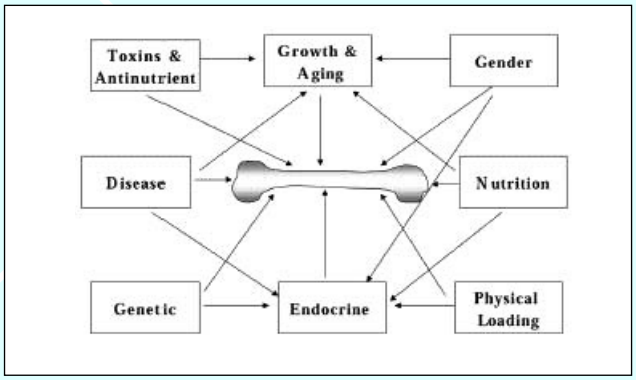


(Source : http://training.seer.cancer.gov/module_anatomy/Unit3_4_bone_classification.html)

Bone Strength

It is the toughness or ability of the bone to endure stress. Bone ash content and bone mineral densities are used as indices of a bones strength. There are various factors affecting a bones strength as depicted in Fig 2.

Fig. 2: Factors affecting bone strength



(Source: Rath *et al*., 2000)

A bone development is controlled by hormones such as parathormone, growth hormone and oestrogen (Rath *et al*., 2000).

The various types of leg weakness/disorders found in poultry are:-

TYPE OF DISORDERS			
I. NON-INFECTIOUS a) Rickets b) Tibial Dyschondroplasia c) Chondrodystrohpy d) Kinky Back* e) Osteopetrosis f) Osteochondrosis g) Femoral Head Necrosis h) Osteoarthrosis i) Rotated Tibia j) Crooked Toes k) Degenerative Joint Disease* l) Spraddle Legs m) Cage Layer Fatigue n) Bone Fractures o) Foot Pad Dermatitis p) Vitamin & Mineral Deficiencies * Occurance is rare	II. INFECTIOUS a) Arthritis & Bumble Foot b) Viral Arthritis c) Mycoplasmosis d) Runting Syndrome e) Synovitis f) Osteomyelitis g) New Castle Disease h) Marek's Disease i) Tick Paralysis* j) Scaly Leg Mite* k) Amyloidosis*	III. POISONING a) Ergotism b) Lathyrosis IV. EMBRYONIC Mineral and Vitamin deficiency in breeder flock	V. RARE AND INHERITED a) Nanomelia* Short bones in homozygotes b) Ametopodia* Absense of metatarrsal and metacarpal bones

I. NON-INFECTIOUS LEG DISORDERS

I. a. RICKETS

It is a developmental disorder noticed in young growing chickens and is characterized by soft poorly mineralized bones and irregular, thickened growth plates. This leads to flexibility in long bones causing lameness (Wise and Nott, 1975). It is attributed to vitamin D_3, calcium (hypocalcemic rickets) or phosphorus (hypophosphatemic rickets) deficieny and/or imbalance. In hypocalcemic rickets, there is a thickening of the epiphyseal plate due to accumulation of proliferating chondrocytes (Jande and Dickson, 1980) with increase in the length of perforating epiphyseal vessels. In hypophosphatemic rickets, there is an accumulation of hypertrophic chondrocytes with poor mineralization (Lacey and Huffer, 1982). In rickets due to Vitamin D_3 deficiency, the growth plate changes are similar to hypocalcemic rickets along with an enlargement of parathyroid and the cells are irregular and vacuolated (Cheville and Horst, 1981). Poor intestinal absorption of calcium and vitamin D_3 can also predispose the birds to rickets in spite of adequate dietary levels (Bar *et al*., 1987).

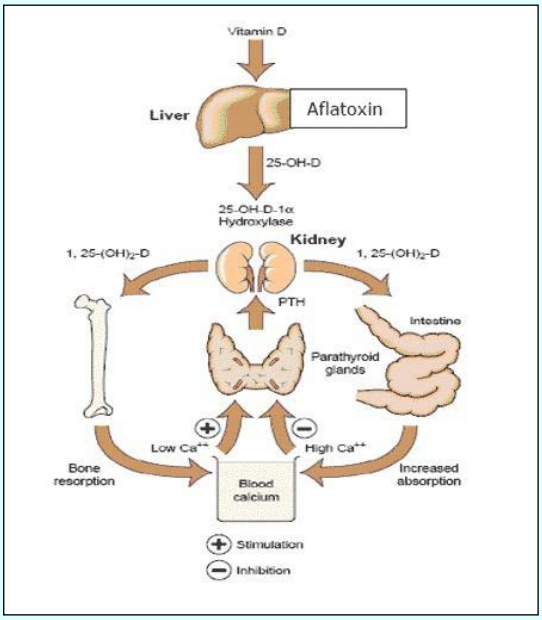
The characteristic features are soft rubbery bones

soft and pliable beak, crooked keels and bowed legs. The rib cage is thickened, flattened and bent. Beading of ribs at the juncture of the spine is also a noticeable sign.

High levels of Vitamin A (> 12000 IU/kg feed) interferes with the vitamin D_3 utilization leading to rickets. Dietary inclusion of rye impairs the fat utilization, leading to poor vitamin D_3 and calcium absorption (Campbell *et al*., 1983). Feeding of tallow causes insoluble calcium soap formation and also interferes with the availability of vitamin D_3. Mycotoxins such as aflatoxin, ochratoxin and fusarium toxin also lead to rickets due to their toxic effects on liver and kidney which consequently prevents the conversion of active form of vitamin D_3 and it's absorption (Verma, 2006) as shown in Fig 3.

(Source: www.myoops.org/.../courses/5/content/215759.htm)

Fig 3: Effect of aflatoxin on Vitamin D absorption



(Source: Verma, 2006)

Rickets can be treated by providing optimum Ca:P ratio, active form of vitamin D_3, addition of vitamin C and top dressing the feed daily with DCP.

I. b. TIBIAL DYSCHONDROPLASIA (TD)

It is a bone developmental disorder seen in 3 to 5 week old fast growing broilers. It is characterized by an abnormal cartilage mass in the proximal head of the tibia and tibiotarsus. The cartilage in the TD growth plate is both unmineralised and avascularised, hence failing to form a bone leading to the retention of thickened plug of dead cartilage. As the bone grows a lateral displacement of the growth plate appears causing characteristic bowing or bending of the legs. The birds are reluctant to move and when forced to walk have a stiff gait.

Various factors can predispose the birds to TD. The incidence of TD varies in response to genetic selection and is related to a major sex linked recessive gene (Leach, 1987). Variation in calcium phosphorus ratio in the form of either low calcium or high phosphorus leads to TD

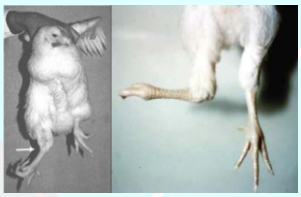
(Paz *et al.,* 2005)

(Edwards, 1984; Riddell and Pass, 1987). Electrolyte imbalance with respect to high chloride levels could predispose birds to TD due to metabolic acidosis which interferes with the metabolism of vitamin D_3 (Luo *et al*., 1992). Presence of thiram in the diet of birds induces lesions similar to TD as thiram interferes with the copper metabolism (Wu *et al*., 1990). High level of cysteine (150-300 ppm) in the diet can cause TD (Bai *et al*., 1994). Mycotoxins in the feed especially *Fusarium mycotoxin* (TDP-1) at 75 ppm causes 100% TD in broilers (Verma, 2006) but not in layers (Wu *et al*., 1993). In leghorn chicks TD is induced by tetramethylthiuram disulfide, an organic fungicide at 30 ppm (Veltman *et al*, 1985). Improperly treated soyabean meal with high urease activity also leads to TD (Edwards, 1985). Reduction in the expression of transforming growth factor-beta also predisposes to this disease. (Thorp, 1994).

TD upto 6% can be prevented by decreasing the growth rate of broilers by following a fasting program of 1 hour each day (Edwards and Sorensen, 1987; Edwards, 2000). Maintaining proper acid base balance, i.e. addition of chloride without balancing for sodium or potassium can lead to metabolic acidosis. High chloride levels can be corrected by adding magnesium to the diet as magnesium plays a role in improving the deposition of copper and zinc in the bone (Luo *et al*., 1992). Usage of active form of vitamin D_3 (1,25 $(OH)_2$ D_3) and casein phosphopeptide (14g/Kg) along with 25 OH D_3 can reduce the incidence of TD in young broiler chickens (Parkinson and Cransberg, 2004). Phytase is effective in reducing incidence and severity of TD in low incidence TD chicks (Punna and Roland, 2001). Copper sulphate supplementation in thiram contaminated diets ameliorates the effects of TD (Wu *et al*., 1990). Similarly inclusion of molybdenum (10 ppm) in high cysteine ration cuts down the TD incidence rate (Bai *et al*., 1994). Petek *et al.* (2005) opined that intermittent lighting (12L:3D) in combination with ascorbic acid (150mg/L in water) was effective in reducing TD in broilers without compromising on the performance. Incidence of TD in breeder flock can be reduced through genetic selection (Hester, 1994).

I. c. CHONDRODYSTROPHY

It is a generalized disorder of the growth plate in long bones in such a way that the linear growth is impaired but the mineralization and appositional growth are normal (Wise and Nott, 1975). It is the major cause of leg problems in broiler chickens affecting birds to varying degrees. It results in short, thickened, misshapen long bones, usually with an enlargement of the hock joint and deviation of the tibiotarsal end. It causes characteristic hock sitting posture with toes directed ventrally in affected birds. It also leads to angular bone deformity either as a knock kneed stance (valgus) or as bowed legs (varus). The angular deformity values of upto 20° are considered normal which doubles in case of deformity (Lynch *et al.*, 1992). Generally males are more affected than females with an incidence of 2-3% in males and 1% incidence per week in older roaster birds after 7 wks of age. The major deformity is in the distal tibiotarsus while the promixal tarsometatarsal may also be affected. In severe cases, the bird walks on the posterior surface of the hock which becomes swollen and bruised, leading to skin penetration of tibia in some cases. The disorder leads to higher culling and downgrading at processing.



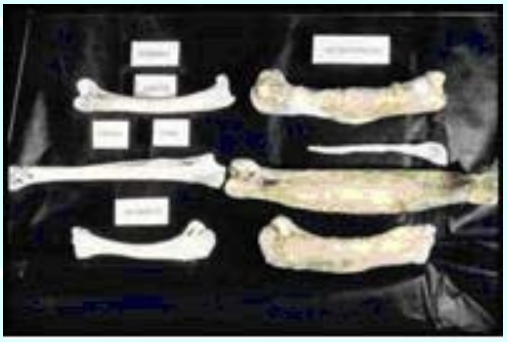
(Source: Rondon, 2008)

Manganese is required for the normal formation of chondroitin sulphate in cartilage and deficiency of the same leads to chondrodystrophy or perosis (Thomas and Lowther, 1976). Inspite of adequate dietary levels of manganese perosis can still be caused due to an excess of dietary phosphorus or calcium. (Halpin and Baker, 1986). Chondrodystrophy is also caused due to deficiency of choline (Lipstein *et al.*, 1977), or zinc as the deficiency interferes with the uptake of sulphur into the epiphyseal growth plate (Riddell, 1975) and other vitamins such as vitamin E, niacin, biotin, folic acid and pyridoxine. Slippery floors can also lead to the angular deformity.

There is no treatment as such after the clinical signs are evident. But, it can be prevented to a great extent by ensuring adequate levels of manganese, choline, zinc, calcium and phosphorus ratio, biotin and other B complex vitamins. Perosis induced by zinc deficiency can be corrected to a certain extent by adding histamine, histidine or aspirin to the diet (Riddell, 1975).

I. d. OSTEOPETROSIS (Marble Bone, Thick Leg Disease)

This disorder arises due to abnormal growth and modeling of bone characterized by thickened legs. The affected bones have a very narrow marrow cavity and thick cortical bone. It can occur either due to deficiency of zinc or due to infection with an avian leucosis complex virus. It can be induced by embryonic or a day old inoculation of chicks with strains of avian leukosis virus (Kirev, 1988) resulting in formation of a highly cellular bone with a marked rise in serum bone alkaline phosphatase activity. It is generally bilateral with the tarsometatarsus and tibiotarsus affected oftenly.



(Source: www.worldpoultry.net)

I. e. OSTEOCHONDROSIS

It is a degenerative leg disorder arising due to disturbance in endochondral ossification causing changes in the epiphyseal or articular cartilage of the bone. It occurs due to focal delay in the replacement of a cartilage by the endochondral bone (Duff, 1990). It is a sequel to TD. The incidence is seen in 15 week old birds and adult broiler breeders where the femur, cervical and thoracic vertebra, hip joint and distal tibiotarsus are the most commonly affected (Duff, 1985; Duff, 1989). The subclinical form of this disease is more prevalent.

I. f. FEMORAL HEAD NECROSIS

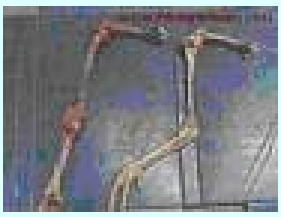
It is a severe degenerative disorder characterized by the head of the femur being separated from the bone shank. It occurs in fast growing birds such as the broiler and turkey. The disorder is seen in 2-4 week old birds and there are no outward signs of the leg deformity except that the birds are reluctant to walk. It arises due to degeneration of the epiphyseal cartilage of the femoral head and subsequent epiphyseolysis (Duff and Randall, 1987). The affected femur is ochre coloured, porous and brittle (brittle bone disease) with the femur head attached to the acetabulum of the hip. It is also associated with scabby hip and in severe conditions can lead to bacterial infection and subsequent downgrading of the carcass during processing. Predisposing factors include immunosuppressive viruses such as IBD virus, CA virus, staphyloccus and non-infectious bone pathologies such as hypophosphatemic rickets.

(Source: McNamee and Symth, 2000)

The disorder can be prevented to some extent by supplementing the diet with Vitamin D_3 or by provision of vitamins via drinking water. The treatment is not effective once the symptoms have set in.

I. g. ROTATED TIBIA

The tibia is twisted during the growth to an extent of 90° or more and is extended laterally. It can be uni or bilateral. The disease has been reported in broilers, turkeys and guinea fowl but is more common in the ostrich and emu causing significant lameness. The disease is seen at 3 weeks of age and the exact etiology is unknown. In turkeys and guinea fowl the disorder may be subsequent to malabsorption syndrome and rickets respectively. It leads to downgrading due to the handling in the processing plants (Riddell, 1981).

(Source: Julian, 1998)

I. h. CROOKED TOES

It is commonly found in broilers and turkeys with toes been bent out of the normal straight alignment. The deformation may be caused by shortening of flexor tendon or hereditary. Faulty management in terms of slippery flooring and infrared brooding can lead to the condition. Pyridoxin deficiency can also lead to crooked toes due to its role in collagen crosslink formation and bone mechanical properties (Masse *et al*., 1996). Overheating of eggs during the first two weeks of incubation also leads to crooked toes.


(Source:www.feathersite.com/Poultry/BRKIncubation.html)
(Source: www.freewebs.com/.../oldenglishgame.htm

I. i. SPRADDLE LEGS

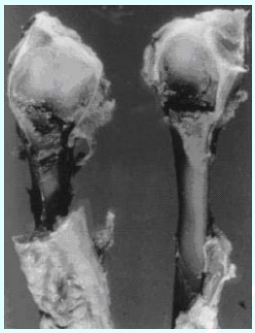
The condition can be uni or bilateral with both the legs splayed laterally from the coxofemoral joint. It is usually due to high humidity during incubation or placing of newly hatched chicks on a slippery floor. Affected birds should be culled (Hester, 1994)

(Source: www.poultryhelp.com/spraddle.html)

I. j. CAGE LAYER FATIGUE

It is the most significant disease seen in laying hens kept in battery cages. The condition is seen in the birds at the peak of egg production and is caused due to inadequate dietary calcium and phosphorus ratio which leads to the erosion of the cortical bone which supplements the mobile calcium medullary reserves. The condition is seen at the peak of egg production i.e. between 25-35 weeks of age when there is considerable amount of osteoclastic activity in the medullary bone due to egg laying. This causes a fracture of the vertebra that subsequently affects the spinal chord. It is seen that at the time of the initial paralysis the birds are apparently healthy. Death occurs due to dehydration or starvation as the affected bird moves to the back area of the cage. The disease in the later stage leads to spontaneous bone fractures during the catching, transporting and processing of the bird. Genetic predisposation to the disease is also suspected. Lack of excercise is seen as another factor that leads to this disease due to the fact that the affected bird recovers once moved to the floor. Provision of perches within the cage environment helps in reducing the incidence of the disease. Also providing adequate dietary levels of calcium during and prior to egg production is essential in preventing this disease, sources of calcium that enable slow release of the mineral such as oyster shell give the best results (Fleming, 2008). Bis-phosphonate, characterized by P-C-P bond has a high affinity for hydroxyapatite and inhibits the resorption of bone and is thus suitable for the prevention of osteoporosis (Thorp *et al*., 1993).

I. k. SPONTANEOUS BONE FRACTURE



(Source: Julian, 1998)

It is the one significant cause of downgrading of poultry carcasses. In old layers the disease occurs as a sequel to cage layer fatigue leading to fractures during catching or transportation. In broilers spontaneous fractures occur during the last part of the growing cycle. Provision of fluorine at 300 ppm in the water can significantly improve the breaking strength and bone ash of younger birds. Again as with cage layer fatigue, bone breakage is more commonly observed in the cage rearing of birds. Balanced nutrition helps in improving the bone strength of birds thus avoiding the occurrence of fractures (Crespo *et al*., 1999)

I. L. FOOT PAD DERMATITIS

It occurs in birds fed biotin deficient diets (Harms and Simpson, 1975). It also occurs due to the faulty litter man-agement. Wet and caked litter stick to the foot pad of the birds leading to cracks in the foot pad which are encrusted with the litter material. This is followed by dryness and flakiness of the skin underneath with abnormal papillary growth (Martland, 1984). In extreme

(Source: McMullian, 1999)

cases the dermatitis becomes hemorrhagic and secondary bacterial infection can occur. The foot pad becomes swollen and ulcerous leading to bumble foot. Inclusion of soyabean meal can also lead to dermatitis due to the excretion of non-digestible oligosaccharides which makes the litter wet and cakey. Feeding adequate quantities of biotin along with proper litter management (litter moisture not more than 25%) aids in the reduced incidence of this disease. Research by Zinpro corporation has shown that broilers fed zinc from Availa Zn have enhanced paw quality and yielded 30% more sellable paws (www.zinpro.com).

I. m. VITAMIN AND MINERAL DISORDERS (Waldenstedt, 2006)

MINERAL/VITAMIN	DEFICIENCY SYMPTOM
Calcium & Phosphorus ratio	Rickets, osteomalacia, osteoporosis
Manganese	Perosis/slipped tendon
Copper	Thin bones and leg weakness
Zinc	Shortened and thickened bones and cartilage, thickening of the scales of limbs (Scaly limb)
Chlorine	Paralysis (Chicks fall forward with their legs outstretched behind them)
Nickel	Enlarged hock joints
Vitamin A	Excess causes leg disorders
Vitamin D	Rickets/cage layer paralysis
Vitamin E	Enlarged and unusually distorted hock
Vitamin C	Required for proper bone formation and bone strength
Vitamin B1	Hock sitting posture
Vitamin B2	Inward Curling of toes and sitting on the hocks (Curled toe paralysis)
Niacin	Enlargement of hock joints and outward bending of the legs
Pantothenic acid	Nodular hyperplasia or cracks in the skin of foot pad and joints of claw
Vitamin B6	Convulsion of legs and wings
Biotin	Dermatitis and Perosis
Choline and Vitamin B12	Perosis

II. INFECTIOUS LEG DISORDERS

II. a. ARTHRITIS AND BUMBLE FOOT

Staphylococcus and E.coli may be the factors leading to hard fibrous swelling with ulceration of the foot pad. Staphylococcal arthiritis along with *Proteus mirabilis* results in pus formation in and around the joints leading to lameness. Reo viral arthritis and *mycoplasma* infection can predispose to this condition.

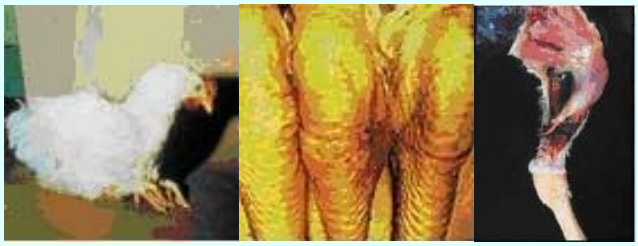
Sporadic cases of arthritis can also be due to *Salmonella spp* and *Streptobacillus moniliformis* (Ross Technical Bulletin, 2001)

(Source:www.worldwidewounds.com/2003/august/Cousquer)

(Vitamin E, B1, Pantothenic acid, B2 and D deficiency, Robinson, APA-ABA youth program)

II. b. VIRAL ARTHRITIS

It is caused by avian Reo virus mainly affecting broiler between 6-7 weeks of age. The disease is mostly asymptomatic or may be accompanied by lameness with the synovial sheath of the tendon of the foot pads or the hock joint inflamed and swollen, ulceration of articular cartilages and haemorrhage in tissues around the articulation (green leg disease). Tenosynovitis is commonly a sequel to viral arthritis. The gastrocnemius tendon of the hock joint can also rupture (Ross Technical Bulletin, 2001).



(Source: www.bpt.com.eg/index_files/REO.htm

II. c. MYCOPLASMA INFECTION

It is caused by *Mycoplasma synoviae* and to some extent by *Mycoplasma gallisepticum* characterized by abnormalities in the joint, reduced walking ability and lameness. Prevention of the infection by effective vaccination and regular serological monitoring of the breeder flock is the only means of controlling mycoplasma infection (Ross Technical Bulletin, 2001).

Source: www.vetserveng.moag.gov.il/NR/rdonlyres

II. d. RUNTING SYNDROME (Helicopter disease, Brittle bone disease, Malabsorption syndrome)

The disease is caused by avian reo virus affecting 1-2 week old birds and may lead to various degrees of leg weakness with lameness and reluctance to move. The broilers partially recover from the disease at about 4 weeks of age but their growth is stunted. The condition predisposes the birds to rickets, TD and valgus-varus deformity (Vertommen *et al*., 1980)

II. e. SYNOVITIS

It may be caused by *Staphylococcus, Salmonella, Pseudomonas* and *E.coli* characterized by swollen joints and tendons with an occasional rupture of the gastrocnemius tendon, thickened capsules, purulent exudates and pitted articular surface. It may get complicated if the primary infection is accompanied by by reovirus or mycoplasma. (Reece, 1992)

II. f. OSTEOMYELITIS

It is characterized by inflammation and infection of the medullary cavity, cortex and periosteum of the bone and is frequently associated with *Staphylococcus spp, Streptococcus spp, E. coli, Proteus spp, Pasteurella spp, Pseudomonas spp, Aspergillus spp* etc. It is predisposed by ischemia, trauma, focal inflammation, bone necrosis and hematogenous spread. The affected birds have lameness, pain, abscessation at the wound site, fever, anorexia and depression. Antimicrobial therapy based on culture and sensitivity is a mandatory treatment for the affected birds (Downloaded from Merck Veterinary Manual).

II. g. NEW CASTLE DISEASE

Lameness associated with New-castle disease virus has been reported in growing pullets (Shirai *et al*., 1986). The ND virus isolated in birds showing leg weakness was 72.2% in broilers and 73.9% in layers with mesogenic strain causing more lameness followed by lentogenic and velogenic strains (Rafique *et al.,* 1997).

II. h. MAREKS DISEASE

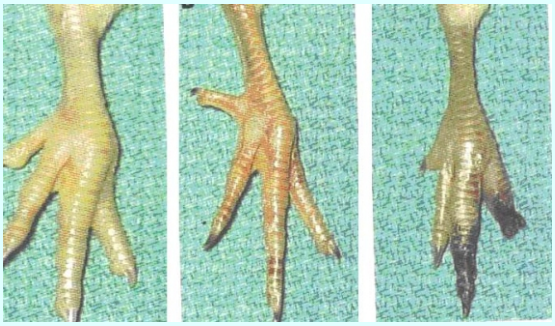
It is a viral disease caused by DNA Herpes virus in birds of 3 months of age or more and may lead to lameness or paralysis of one or both the legs-sportsman posture (Morgan *et al*., 1991).

(Source: The Merck Veterinary Manual)

III. LEG DISORDERS DUE TO POISONING/ ANTINUTRIENTS

III. a. ERGOTISM

It is caused by ingestion of cereal or grass grains infected by the fungus *Claviceps purpurea*. The clinical signs are blackening of beaks, claws and feet followed by severe necrosis and gangrene at the tip of the toes. Chicken feed should not contain more than 0.3% of ergot (Wojnarowicz, 2006).



(Source: Wojnarowicz, 2006)

III. b. LATHYROSIS

Certain sweet peas contain lathyritic agent, β-aminopropionitrile that inhibits the enzyme lysyl oxidase, preventing crosslinks and mineralization consequently decreasing the bone strength (Seigel and Fu, 1976).

IV. DISORDERS IN THE EMBRYO

Folic acid deficiency in the breeder parent leads to distorted limbs in embryo. Biotin deficiency causes embryonic death in the first week or the last 2-3 days of incubation characterized by parrot beak, distorted legs and web formation between 3rd and 4th phalanges. Embryonic death due to inadequate vitamin B_{12} around the 17th day of incubation leads to embryos with atrophied leg muscles. Manganese deficiency causes osteodystrophy and subsequent death in embryos during 20-21 days of incubation. Deficiency of zinc in the breeder flock leads to deformed bones (short and thickened bones) in the embryo. Selenium deficiency causes atrophy or deformity in the embryos with the absence of upper beak, eyes and limbs (Saif *et al*., 2003).

Eggs held for too long before being set in the incubator increases the number of crippled and weak chicks. Low humidity in the hatcher makes the chick more prone to crippling and other leg problems (Goan, 2005).

CONTROL OF LEG DISORDERS

Complexities of factors determine the control of various forms of leg disorders. Control can be effective in two stages. Stage 1 involves the management and control of leg disorders as they occur in the farm. Stage 2 involves the development and implementation of strategies for future prevention and control (Thorp, 1994)

a. GENETICS

Selection for rapid growth rate should be such that there is a proportional increase in the muscle mass along with the skeletal mass. Leg weakness that have genetic predisposition should be taken care of and birds with visible disorders should not be used for breeding and should be culled. Study by Kestin *et al*. (2001) revealed that the lameness in modern genotype of broilers is a result of their selection for high live weights and rapid growth rates, leading to an abnormally high load being placed on relatively immature bones and joints. Kestin *et al*. (1999) conducted a study to investigate the susceptibility of different genotype of broilers to leg weakness and found that Ross had the lowest score for leg weakness and the cobb birds had the highest score. Similarly the gait score was the best with Ross and the worst with Cobb.

b. NUTRITION

Adequate mineral and vitamin nutrition with optimal calorie protein ratio should be ensured to alleviate the leg problems. A well balanced diet is essential in breeders to prevent leg disorders in the progeny. High nutrient dense diets and diets high in protein energy can also predispose the birds to leg problems. Enzymes should be regularly used in the diet for better utilization of nutrients which enhances bird performance and litter quality. Feed ingredients which result in poor litter quality are often associated with an increase in leg weakness. Sorghum and rapeseed meal to a certain extent induce leg weakness due to presence of tannin or phytate in these ingredients. Similarly a soyabean meal with high trypsin inhibitor activity can lead to leg abnormalities. Usage of dietary fats with high free fatty acid levels interfere with the mineral absorption and availability and might induce leg problems. Hence, proper selection of feed ingredients is a must (Ross Technical Bulletin, 2001).

c. MANAGEMENT

The following points should be taken care of to reduce the incidence of leg disorders

1. Proper brooding temperature
2. Proper hatchery management
3. Proper ventilation
4. Litter quality
5. Enough feeder and drinker space
6. Proper exercise
7. Supplementation of vitamins in drinking water (Vitamin D, C and B-complex)
8. Timely vaccination
9. Adequate floor space with less stocking density
10. Usage of perches in cages
11. Fasting or restricted feed programs and lighting pattern to reduce rapid growth
12. Proper handling of birds

d. ENVIRONMENT

d. 1. LIGHTING PROGRAM

Constant light promotes rapid growth but leads to more problems like lower activity and leg disorders (Manser, 1996). Intermittent lighting aims to reduce the early growth by providing 4-9 hours of darkness and dim light during the daily light period after 1 week of age. During the last two weeks prior to processing the light intensity is again increased to stimulate growth. Highest gait score was recorded in males and females provided with 20 L: 4D (Brickett *et al*., 2007). Use of fluorescent bulbs cause a lower incidence of leg problems as compared to incandescent bulbs (Lewis and Morris, 1998).

d. 2. ENVIRONMENTAL TEMPERATURE

Both high and low temperature is associated with increased incidence of leg disorders (Hulan and Proudfoot, 1987). The birds must be reared in their optimum thermoneutral zone.

CONCLUSION

As detailed above, leg weakness/disorders covers a wide range of abnormalities due to a multitude of etiological causes. It has become one of the most prevalent causes of culling and late mortality in fast growing broilers and high producing layers. They have a severe impact on the welfare of birds causing pain and impairing their gait pattern leading to reduced walking ability. It ultimately affects the growth and production performance of the birds consequently causing a huge economic loss to the producer. It also leads to condemnations and carcass downgrading at the processing plant. The incidence of each leg disorder varies from farm to farm. Leg disorders can be prevented by modifying the bird's environment and diet, so as to slow the growth of the birds. Growth rates can be reduced by altering the artificial lighting regime and restricted feeding practices. Adequate balanced ration with optimum mineral, vitamin and calorie protein ratio should be fed to the birds. Care should be taken to avoid mineral imbalances. Phytase should be added to the diet to avoid mineral antagonisms. Adequate mineral and vitamin nutrition in the breeder flock is essential to prevent disorders in the embryo. Also, proper managemental practices should be adopted. For disorders having genetic predisposition, genetic selection against the same should be carried out.

References are available with author and can be made available on request.

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