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Review

Nutritional factors of importance for optimal leg health in broilers: A review☆

Lotta Waldenstedt*

Swedish University of Agricultural Sciences, Department of Animal Nutrition and Management, Avian Division, Funbo Lövsta Research Centre, SE-753 23 Uppsala, Sweden

Abstract

Leg disorders in broilers are important welfare and economic issues for the poultry industry. Common problems are tibial dyschondroplasia, angular bone deformities, *i.e.* lateral or medial deviation of the distal tibia or proximal metatarsus, and rickets. Although many of the problems can be reduced or eliminated by slower growth rate, this does not necessarily mean that rapid growth and heavy birds will result in leg disorders, as these problems are today generally more related to interactions between genetic, management and nutritional factors. The objective of the current paper is to provide an overview of nutritional factors that influence leg abnormalities in broiler chickens. Deficiencies and excesses of vitamins, minerals and minor nutrients may affect leg health. Nutrients of major concern are Vitamin D, calcium and phosphorus. Other vitamins, such as A, E, C, folacin and several B-vitamins are also of importance. Further, a number of minor elements, which have been shown to influence leg disorders, either in the field or under experimental conditions, are reviewed. Complex interactions between various nutrients may further complicate formulation of broiler diets for optimal leg health.

Keywords: Poultry; Nutrition; Skeletal disorders; Leg weakness; Lameness

Abbreviations: TD, tibial dyschondroplasia; 25-OHD, 25-dihydroxycholecalciferol; 1-25-OHD, 1,25-dihydroxycholecalciferol

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* Tel.: +46 18 67 4531; fax: +46 18 67 2946.
E-mail address: Lotta.Waldenstedt@huv.slu.se.

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Contents

1. Introduction

Leg disorders in broilers are important welfare and economic issues for the poultry industry. Mortality, number of culls and condemnations due to leg abnormalities have been estimated to cause 0.10–0.30 of the total loss (Julian, 1995). Leg disorders and bird mobility are affected by a number of different bone, tissue and neural disorders, as well as by other factors. Deficiencies, and occasionally excesses, of certain essential nutrients play major roles in the development of several leg abnormalities in poultry, even though many leg and mobility disorders are outside the influence of nutritional factors. Among the most common leg abnormalities affected by nutritional practices are tibial dyschondroplasia, resulting from inadequate vascularisation and ossification of the growth plate resulting in an abnormal mass of cartilage under the growth plate, causing unnatural biomechanical forces, and therefore gait alteration, additional bone abnormalities, and even fractures (Julian, 1998; Farquharson and Jeffries, 2000). Angular deformities of the long bones, such as varus–valgus deformities and "twisted legs" caused by a lateral or medial deviation of the distal tibia or proximal metatarsus, frequently accompanied by slippage of the gastrocnemius tendon are other common leg disorders (Riddell, 1992). Also rickets, characterized by an enlargement of the epiphyseal area and occasionally deformity of the soft, rubbery bone (Julian, 1998), does occasionally occur due to nutrient deficiencies and/or imbalances.

Cartilage abnormalities can also act as foci for bacterial infections resulting in the severe welfare problems of osteomyelitis, an infection of the proximal part of the tibia or femur (sometimes called femoral head necrosis), usually with *Staphylococcus*, although other infectious organisms may also be present (Butterworth, 1999; McNamee and Smyth, 2000). Infectious diseases in skeleton, joints and soft tissues, as well as infections of the central and peripheral nervous system may also be causes of leg abnormalities, but these are generally not influenced by nutritional factors.

The aetiology of leg abnormalities is generally complex and not outwardly related to a single factor, and there is sometimes an overlap between the aetiology, pathology and clinical signs of these conditions. Factors that affect the intestinal epithelium, and thus decrease nutrient absorption, *e.g.* bacterial, viral and parasitic infections, as well as antinutritional factors in the feed, may also induce leg disorders caused by nutrient imbalances. Thus, genetics, management, nutrition, hygiene, and concurrent diseases will influence the occurrence of leg disorders, both under field and experimental conditions. Consequently, even when diet contents appear to be adequate, leg abnormalities may occur in a flock. Leg disorders are often accompanied by uneven growth in the flock, even though a high incidence of leg abnormalities may be present in a flock with little or no signs of reduced weight gain or feed utilization (Summers et al., 1984). Further, rickets and TD can cause distortions of bone growth that may not be apparent at the time of the deficiency but may show up later in the growing period and result in clinically manifest leg bone abnormality, even though the bird by then is receiving an adequate diet (Whitehead et al., 2004). Thus, post mortal examinations of the skeletal, muscular and nervous systems, in addition to feed analyses, are important for correct diagnoses of leg disorders in the field. However, frequent failures to find explanations of leg problems at necropsy, or the opposite, the finding of skeletal and/or cartilaginous lesions in the absence of clinical manifestation in broilers further stresses the complexity of the problem.

The aim of this paper is to give an overview of nutritional factors that are important to consider when formulating broiler diets for optimal leg health. Over the past 40–50 years, numerous research reports have been presented on the subject. However, sometimes it is uncertain if the results are still valid for modern broiler genotypes reared with well-balanced commercial feeding and good management practices. Many of the leg disorders in broilers described from the field and in research reports today are not synonymous with the early perotic conditions often reported in older studies. References to past literature are sometimes confusing owing to several distinct entities being given the same name: perosis for example (Nairn and Watson, 1972; Wise, 1989 and Whitehead, 2002a). Nowadays, the term chondrodystrophy is generally usually used to replace perosis (Wise, 1975; Thorp, 1992). Chondrodystrophy is defined as a generalized disorder of the growth plates of long bones such that growth is impaired, while mineralisation and appositional growth remain normal, resulting in shortened long bones, enlargement of hock joints and often secondary valgus or varus deformations. In severe cases, the gastrocnemius tendon may also become displaced from the medial condyle (Riddell, 1992). Generally, chondrodystrophy seems not to be a problem in broiler flocks today (Engström et al., 2003), even though the role of minor chondrodystrophies in the development of bone deformities is not fully known (Thorp, 1992).

2. Vitamins

Broiler diets are routinely supplemented with minerals and vitamins within ranges designed to avoid deficiencies or toxicities. The amounts required have been established experimentally and are reviewed regularly (*e.g.* NRC, 1994). However, these requirement values are the minimum needed under good experimental conditions. In commercial diets

Table 1
Broiler requirements (per kg diet) of vitamins important in bone formation (NRC, 1994; Whitehead, 2002b)

	NRC		Whitehead	
	0–3 weeks	3–6 weeks	Starter	Finisher
Fat soluble vitamins				
Vitamin A (IU)	1500	1500	1400	1400
Vitamin D_3 (IU)	200	200	800	800
Vitamin E (IU)	10	10	10	10
Water soluble vitamins				
Biotin (mg)	0.15	0.15	0.18	0.18
Choline (mg)	1300	1300	1300	1300
Folacin (mg)	0.55	0.55	2.5	2
Niacin (mg)	35	30	70	70
Pyridoxine (B_6) (mg)	3.5	3.5	4	4
Riboflavin (B_2) (mg)	3.6	3.6	5	5

IU Vitamin A = 0.3 μg retinal; IU Vitamin D_3 = 0.025 μg cholecalciferol.

most vitamins are generally added in excess to meet the birds' higher requirements during more stressful conditions encountered in practical rearing, as well as to account for losses occurring during diet preparation and storage. Recommendations of vitamins important to bone formation in broilers are given in Table 1.

2.1. Vitamin D

Most raw materials in broiler diets contain little or no Vitamin D, therefore, this vitamin is generally added to the diet in synthetic form. As Vitamin D_2 (ergocalciferol) has only about 0.1 of the potency of D_3 for poultry, Vitamin D_3 (cholecalciferol) is used (McDonald et al., 1995). The quantitative requirement of young growing chickens for Vitamin D_3 is usually based on measurements of bone quality, such as bone ash content or incidence of rickets, which have generally been found to be more sensitive indicators than growth rate (Edwards et al., 1994). The current NRC (1994) requirement for chicks between 0 and 3 weeks of age when diets contain adequate levels of calcium and phosphorus is 5.0 μg/kg diet. However, in commercial practice, diets are routinely fortified with supplemental Vitamin D_3 in the range 75–125 μg/kg (Whitehead et al., 2004). Additionally, water-soluble vitamin mixtures containing Vitamin D_3 may be added during the first few days of life as a means of minimizing early growth problems.

Recently, Whitehead et al. (2004) showed that Vitamin D_3 requirements of broilers up to 14 days of age at sufficient dietary calcium and available phosphorus concentrations may be in the range 35–50 μg/kg of feed for cortical bone quality. After 14 days, the Vitamin D_3 requirement for cortical bone quality decreases to less than 20 μg/kg of feed. These requirements are much higher than earlier estimates and may be related to higher calcium requirements of modern broiler genotypes. Rickets due to Vitamin D_3 deficiency seem to be eliminated at approximately 40–45 μg/kg of feed (Ledwaba and Roberson, 2003), even though it has been reported that doses as high as 77.5 μg cholecalciferol/kg have been needed to eliminate rickets (Elliot and Edwards, 1997).

The study by Whitehead et al. (2004) also showed that 250 μg cholecalciferol/kg of feed had a preventive effect on TD. Vitamin D_3 has previously not been reported to be associated with prevention of TD, although Vitamin D_3 metabolites have been shown to be very effective in that role. In most other studies, however, much lower Vitamin D_3 concentrations (normally within the range 25–75 μg/kg) were used, which could explain the lack of response. High levels of cholecalciferol have also been shown to increase the utilization of phytate phosphorus and the retention of calcium and phosphorus (Mohammed et al., 1991).

A further factor that may influence Vitamin D_3 requirements is the dietary calcium and phosphorus. An increase in calcium and phosphorus requirements in modern broiler genotypes will thus influence Vitamin D_3 requirements. In 3-week-old broilers fed a diet deficient in phosphorus, very high levels of cholecalciferol (1250 μg/kg of diet) were required to produce maximum growth and bone ash (Baker et al., 1998). In the same study, however, when the diet was slightly deficient in calcium, or was adequate in both calcium and phosphorus, adding more than 30–40 μg cholecalciferol/kg of diet produced no response on growth, feed efficiency or bone ash values. It should be noted that ultraviolet light also influences the Vitamin D requirement of the birds, especially during cholecalciferol-deficient diets, but this is usually not an issue in intensive broiler production where birds are reared in enclosed houses.

Excess Vitamin D_3 in the diet may have a negative effect on leg health and body weight. In the study by Baker et al. (1998), young broilers at second and third week of life tolerated very high Vitamin D_3 concentrations, up to 1250 μg/kg, as measured by bone ash and body weight gain. However, older studies (Lofton and Soares, 1986) showed an increase in the incidence of leg abnormalities and particularly TD as the Vitamin D_3 level increased up to 500 μg/kg. Also Cruickshank and Sim (1987) found an increased incidence of varus and valgus deformities, as well as a slight decrease in body weight gain at 100 μg Vitamin D_3 per kg. At what levels impaired leg health and performance due to excess Vitamin D_3 would occur in modern broilers, with their obviously higher Vitamin D requirements, is not completely clear.

2.1.1. Vitamin D metabolites

Vitamin D metabolism in the bird is a complex process involving several metabolites. Dietary Vitamins D_2 and D_3 are absorbed through the small intestine and are transported in the blood to the liver where they are converted into 25-hydroxycholecalciferol (25-OHD), the major circulating form of Vitamin D_3. 25-OHD is then transported to the kidneys where it is converted into 1,25-dihydroxycholecalciferol (calcitriol) (1,25-OHD), which is the most biologically active, hormonal metabolite of the vitamin (McDonald et al., 1995). Stressful conditions, such as high bird density, heat stress, mycotoxicosis, enteritis, malabsorption syndromes, and certain immune disorders may impair absorption or liver hydroxylation of cholecalciferol, which is one rationale for the use of Vitamin D metabolites in broiler feed. Moreover, as the current legal dietary limit in the EU is 125 μg cholecalciferol/kg, the feeding of Vitamin D_3 metabolites may be a means of providing the high Vitamin D_3 status needed by young broilers (Whitehead et al., 2004).

The biological activity of 25-OHD is estimated to be 2–2.5-fold that of cholecalciferol (Boris et al., 1977; Edwards et al., 1994; Ledwaba and Roberson, 2003). Supplementing

the diet with 25-OHD has been reported to cause a significant reduction in the incidence and severity of TD, even though the effects are not of the magnitude or consistency as those effects caused by 1,25-OHD supplementation (Rennie and Whitehead, 1996; Mitchell et al., 1997; Zhang et al., 1997; Edwards, 1989; Roberson, 1999). In those studies showing a preventative effect of 25-OHD on TD, the supplement was added to the diet at levels between 75 and 344.5 μg/kg. Further, there seems to be an interactive effect with calcium levels in the feed. At low calcium levels, 25-OHD seems to be more effective in reducing TD incidence, than at sufficient calcium levels (Ledwaba and Roberson, 2003). In the same study, for chickens exposed to UV-light, overall TD incidence was minimized at 10 μg 25-OHD/kg feed. Concentrations of up to 690 μg 25-OHD/kg have been given to chickens without adverse effects on growth rate or feed efficiency (Yarger et al., 1995a,b).

Also dietary supplement of the metabolite 1,25-OHD within the range 5–10 μg/kg feed has in many studies been reported to alleviate the incidence and severity of TD and rickets, and increase bone mineralisation, both in diets adequate in calcium and cholecalciferol, and at suboptimal levels of calcium and phosphorus, even though results are not completely conclusive (Edwards et al., 1992; Rennie et al., 1993, 1995; Thorp et al., 1993; Roberson and Edwards, 1994; Elliot and Edwards, 1997). Supplement of 1,25-OHD with 3–6 μg/kg feed showed no negative effect on growth when diets contained low dietary calcium concentrations, but as dietary calcium concentrations approached 10 g/kg growth was depressed (Elliot et al., 1995; Rennie et al., 1995; Roberson and Edwards, 1996).

In experimental studies, several other Vitamin D metabolites, such as 1,24R,25-trihydroxycholecalciferol, 1,25-dihydroxycholecalciferol-dihydroxy-26,27-hexadenteniumcholecalciferol, and 1,25-dihydroxycholecalciferol-dihydroxy-24R-fluorocholecalciferol have also been shown to be effective in preventing the development of TD in broilers (Edwards, 1990).

2.2. *Vitamins A and E*

Vitamin A-deficient birds have poor calcification and bone development (Thompson et al., 1967). However, deficiencies are rare in modern broilers, as this vitamin is generally added to the diet at relatively high levels. Effects of excessive levels of dietary Vitamin A (retinol) on the incidence of TD in broilers is not conclusive; some studies indicate that high levels may increase the incidence of TD, whereas, others show no response, or even a decrease of TD-incidence at high Vitamin A levels (Jensen et al., 1983; Veltmann et al., 1986; Aburto and Britton, 1998a,b; Aburto et al., 1998; Ballard and Edwards, 1988; Whitehead et al., 2004). The severity of Vitamin A hypervitaminosis is affected by nutrient interactions, *e.g.* interactions with other fat-soluble vitamins (Veltmann et al., 1986; Aburto and Britton, 1998b). However, as shown by Aburto and Britton (1998b), high dietary levels of Vitamin A negatively affected the utilization of Vitamin D_3 only when this vitamin was present at a marginal level (500 IU/kg) in the diet. At Vitamin A ranges likely encountered in usual commercial practice (8000–15,000 IU/kg), Whitehead et al. (2004) found no indication of interaction between dietary Vitamins A and D_3 levels.

A deficiency of Vitamin E leads to an increased incidence of leg abnormalities, especially lateral or medial deviation of the distal tibia or proximal metatarsus (Summers et al., 1984). Vitamin E deficiency may also lead to muscular dystrophy manifested in impaired mobility

(Austic and Scott, 1991). The synergistic effect of Vitamin E and selenium also needs to be considered in this context. High dietary levels of Vitamin E negatively affected the utilization of Vitamin D_3 in chickens fed either calcium-deficient, or Vitamin D-deficient diets (March et al., 1973; Murphy et al., 1981; Aburto and Britton, 1998a).

2.3. B-vitamins

Deficiencies of a number of B vitamins have been reported to result in leg abnormalities. In particular, pyridoxine (Vitamin B_6) deficiency has been associated with stunted longitudinal bone growth, and supplementation has been shown to reduce the incidence (Masse et al., 1996). Pyridoxine may exert its beneficial effect via involvement with zinc homeostasis. The metabolic process is further complicated through the interaction of dietary protein, as a high level of dietary protein presumably increases the metabolic requirement for pyridoxine through processes as transamination and/or deamination (Daghir, 1976).

There is generally low availability of biotin in grains such as barley and wheat, and in some older studies biotin deficiency has been associated with deformity of the tibio-metatarsal joint frequently accompanied by slipped tendons, even though other studies found no such effect (Roland and Edwards, 1971; Whitehead et al., 1976). Footpad dermatitis, which may be associated with biotin deficiency (Harms et al., 1977), has also been suggested as a contributing factor for bacterial infections spreading to the joints.

A deficiency of choline is associated with deformity of the tibio-metatarsal joint frequently accompanied by slipped tendons (Pesti et al., 1981; Stock and Latshaw, 1981), and TD (Ferguson et al., 1978; Summers et al., 1978). Deficiency of niacin (nicotinic acid) gave a high percentage of leg deformities, especially associated with medial deviations of the tibio-tarsal and tarsal-metatarsal bones as shown by Summers et al. (1984). Corn-soybean diets in particular are low in niacin (Ruiz et al., 1990). Also riboflavin (Vitamin B_2) deficiency is associated with leg deformities and particularly curled-toe paralysis, a specific symptom caused by peripheral nerve degeneration, in which the chicks walk on their hocks with the toes curled inwards (Gries and Scott, 1972; Summers et al., 1984; Jortner et al., 1987).

Slinger and Pepper (1954) showed that feeding antibiotics to turkey poults prevented leg abnormalities induced by biotin or pantothenic acid deficiencies. Both penicillin and terramycin were found to reduce the requirements of the poult for these vitamins, and this was also found to be true for niacin (Slinger et al., 1953). A partial explanation for the "sparing" effects of antibiotics on the requirement of the poult, as suggested by the authors, is the reduction in the numbers of intestinal micro-organisms, which compete with the host for B vitamins, giving a higher availability of these vitamins to the chicken. However, it is unclear whether this effect is of practical significance in modern broiler feeding. In the light of the new EU legislation prohibiting the use of antibiotic growth promoters this may, however, warrant further considerations on the requirement of these B vitamins for optimal leg health in modern broiler genotypes.

2.4. Vitamin C

Vitamin C (ascorbic acid) is involved in biosynthesis of 1,25-OHD and collagen, and dietary supplementation with ascorbic acid results in significant effects on development of

TD in some animal experiments, but not in others, and it is unclear under exactly what conditions this relationship is important in practical prevention of TD (Weiser et al., 1988; Foulds, 1993; Whitehead et al., 1994; Rennie and Whitehead, 1996).

2.5. *Folacin*

Folacin (folic acid) deficiency may result in increased frequencies of lateral or medial bone deviation with slipped tendons, and crooked toes (Summers et al., 1984). Diets high in protein and fat can interfere with folic acid metabolism, which may increase leg disorders at marginal folic acid levels (Creek and Vasaitis, 1962; Wong et al., 1977; Ryu et al., 1995).

3. Calcium and phosphorus

Calcium and phosphorus are the two most abundant minerals in bone constituting approximately 370 and 170 g/kg bone ash, respectively (Doyle, 1979). Bone is a highly complex structure, and the composition varies according to the age and nutritional status of the animal. As the skeleton is not a stable unit in the chemical sense, the exchange of calcium and phosphorus between bones and soft tissue is a continuous process. The balance between these nutrients is important, since an abnormal ratio may be as harmful as a deficiency of either element in the diet. The main consequence of suboptimal levels of these minerals is rickets, either of the calcium deficiency or phosphorus deficiency type, which can occur when the dietary content of either nutrient is too low, or the dietary content of one is too high and induces a deficiency of the other. An increase in the incidence of TD is another consequence of an imbalance when the calcium:phosphorus ratio is suboptimal (Kling, 1985). TD can still occur even under optimum calcium and phosphorus feeding, but rickets should be preventable by correct diet formulation. Given the rigidity of dietary specifications for calcium and phosphorus, and the importance of avoiding excessive use of phosphorus to minimize pollution, dietary contents sometimes fail to meet specifications (Whitehead et al., 2004).

Broiler diets generally contain extra supplies of calcium and phosphorous. Calcium has generally a relatively high availability from most sources, but the availability of phosphorus varies largely depending on the source, and considerable attention has been paid to the availability of this element. Much of the phosphorus in cereal grains is in the form of phytates, which have low availability for the chicken, and therefore mainly non-phytate sources are considered when formulating diets. The use of phytase further complicates the subject of phosphorus utilization and metabolism in broilers, and in some cases phosphorus availability may be lower than expected, especially at those marginal phosphorous levels used in order to minimize excretion. In a study by Scheideler and Ferkert (2000), phytase supplementation of a diet containing either 3.0 or 4.5 g non-phytate phosphorus/kg of diet decreased TD incidence. As phytase activity will affect both calcium and phytate phosphorus utilization (Sebastian et al., 1998), there is a need to further evaluate the impact of phytase on leg disorders in broilers.

Non-infectious factors which may affect gastrointestinal absorption of calcium and phosphorus include dietary concentration and physical and chemical forms of these minerals,

passage rate of feed and viscosity of digesta, chelating agents and mineral interactions, gastrointestinal tract pH, and interactions with dietary protein, fat and carbohydrate (Whitehead et al., 1971; Dewar, 1986; van der Aar et al., 1983; van der Klis, 1993; Sebastian et al., 1998).

For calcium, optimal requirements for bone calcification are higher than those for body weight gain, but for phosphorus, requirements for growth and bone mineralization seem similar (Bar et al., 2003). The calcium:phosphorus ratio given to poultry is generally within the range 1:1–2:1. A normal content of starter diets is about 10 g calcium and 4.5 g available phosphorus/kg feed, an approximate ratio of 2:1. However, Williams et al. (2000a,b) showed that in modern broilers, calcium:phosphorus ratios in the bone up to 11 days was up to 2.6:1, and that dietary contents of 12 g calcium and 4.5 g available phosphorus/kg feed give the most normal growth plate morphology at 2 weeks of age.

4. Minor nutrients

Apart from many vitamins and minerals, deficiencies or excesses of several minor nutrient elements can also influence bone development. A number of studies relating to deficiencies of trace minerals associated with leg disorders have been published throughout the years (for a review see *e.g.* Edwards, 1992).

Dietary imbalance between sodium, potassium and chloride may induce leg abnormalities, especially TD (Mongin and Saveur, 1977; Hulan et al., 1987). Increasing the chloride content of the diet markedly enhance cartilage abnormalities, especially when the sodium and potassium content of the diet is low. A possible explanation for this could be that with excess chloride, chicks become acidotic, and there are indications that acidotic chicks may have a decreased capacity to synthesize 1,25-OHD (Sauveur et al., 1977). Thus, if the diet is high in chloride, it must be balanced with equimolar concentrations of sodium and potassium in the form of readily metabolizable anions. However, an increase in potassium and sodium may affect water intake and litter condition, which must also be considered.

Also a number of other elements have been shown to influence leg disorders in broilers. The effect of manganese deficiency on the development of the growth plate and displacement of the gastrocnemius tendon is well documented, and some evidence suggests that interactions with high dietary phosphorus and calcium levels may be a complicating factor (Wedekind and Baker, 1990a,b). Further, deficiencies of zinc (Young et al., 1958), boron (Hunt and Nielsen, 1981; Kurtoglu et al., 2005), copper (Leach and Nesheim, 1965), fluoride (Merkley, 1976), silicon (Carlisle, 1980) and vanadium (cited in Edwards (1992, p. 182)) have been associated with leg abnormalities in growing chickens, at least during experimental conditions.

Some metal elements (*e.g.* magnesium, zinc, strontium, lead, and aluminium) can be incorporated in bone formation processes in place of calcium in amounts depending upon the circulating concentrations (*i.e.* low calcium or high element concentrations will increase incorporation), and toxicities can induce leg abnormalities (Whitehead, 2002a).

Recommendations of essential minor nutrients important in bone formation is given in Table 2. The birds' requirement of minor nutrients are usually well covered in conventional commercial broiler diets, both as natural components of the ingredients and through addition

Table 2
Broiler requirements (per kg diet) of essential minor nutrients important in bone formation (NRC, 1994)

	0–3 weeks	3–6 weeks
Chloride (g)	2	1.5
Magnesium (mg)	600	600
Manganese (mg)	60	60
Potassium	3	3
Sodium	2	1.5
Zinc (mg)	40	40

of trace element premixes. Single minor nutrient elements usually have little impact upon bone characteristics under normal practical nutritional conditions, even though complex interactions between these and other nutrients may be of importance. However, the subject of minor nutrient interactions is extensive and outside the scope of this review.

5. Growth

As leg disorders are more prevalent in broilers and turkeys than in egg-type birds, and many skeletal defects in broilers are rare or absent in slower growing strains (Havenstein et al., 1994; Kestin et al., 2001), growth rate and/or body weight seem obvious causative factors. Although many of the problems can be reduced or eliminated by slowing growth rate, rapid growth and high body weight do not necessarily result in leg disorders.

Reducing growth rate by decreasing feed intake and/or nutrient densities is not easily compatible with welfare demands. There are numerous reports of general nutritional factors influencing growth rate. For example, it has been suggested that energy restriction in the first few weeks of growth reduces leg disorders in broilers (Hulan and Proudfoot, 1987). Also a lowered protein intake that reduces bird growth rate has been shown to result in fewer leg abnormalities (Hulan et al., 1980).

Another way of reducing growth is by restricting feed intake. The length of time each day that feed is available to young broiler chickens significantly influences the development of skeletal abnormalities (Edwards and Sorensen, 1987; Elliot and Edwards, 1994; Su et al., 1999). Two- or 4-h feed deprivation did not significantly reduce the development of TD, whereas, 8 or 10 h deprivation during the night or day was effective in reducing TD (Roberson et al., 1993; Elliot and Edwards, 1994). Also meal feeding treatments (two, three, or four times per day) reduced TD and gait scores as compared to birds fed *ad libitum* (Su et al., 1999). Effects of feed restrictions seem not, however, solely to depend on a restriction in growth, but could also reflect altered feeding patters, increased overall activity, as well as changes in the pattern of circulating hormones such as insulin and growth hormones (Edwards and Sorensen, 1987; Buyse et al., 1997; Edwards, 2000).

Many management factors, such as lighting programmes, can of course also reduce leg disorders, mainly by decreasing early growth (Classen and Riddell, 1989; Gordon, 1994). However, leg abnormalities today are most likely related to more complex situations than only growth *per se*, including interactions between genetic, management and nutritional factors.

6. Other factors

It is generally agreed that leg disorders may be influenced by feed ingredient type and source. For example, rapeseed meal and sorghum, as well as the source of soy bean, have been suggested to influence the occurrence of TD, most likely due to high levels of tannins, although the mechanisms involved are not completely clear (Armstrong et al., 1973; Seth and Clandinin, 1973). However, more recent publications in the scientific literature on the subject are scarce. Other antinutritional factors which are likely to influence leg abnormalities via direct or reduced feed intake effects are lectins, glucosinolates, alkaloids and saponins (Foulds, 1993). Diet dilution with whole grains (*i.e.* wheat), as is common practice in some countries, will reduce the absolute nutrient intake, and it is important to ensure that daily intakes of critical nutrients are satisfied.

Feedstuffs contaminated with certain mycotoxins can induce or aggravate skeletal problems. Aflatoxin and ochratoxin both decrease bone strength because of interference with Vitamin D metabolism, and grains contaminated with *Fusarium* or *Aspergillus* species have been shown to increase the incidence of bowed and twisted legs and TD (Sharby et al., 1973; Haynes et al., 1985; Krogh et al., 1989; van Wel et al., 1993). In practice, higher dietary levels of Vitamin D are often fed when mycotoxins are thought to interfere with calcium absorption and bone formation. Fungicides used in grain treatment can lead to an increased incidence of TD in chickens when they are given contaminated feed (Vargas et al., 1983). Further, the ionophore salinomycin has been shown to prevent cartilage degradation *in vitro*, which would predispose for TD. However, concurrent studies *in vivo* showed no effect on TD incidence, even at high doses of the ionophore (Peters et al., 2002). The antibiotic growth promoter zinc bacitracin gave an increase in TD incidence under experimental conditions (Stub and Vestergaard, 2001).

Some leg abnormalities such as TD are to some degree inherited (Sheridan et al., 1978). Thus, under practical conditions differences in leg abnormalities can be found between hybrids in response to selection applied by different breeding companies. Variability and differences between experimental results may thus in some cases partly be attributed to the broiler strain used (Hulan et al., 1980). In addition to the confounding effect of genetics on skeletal development, there seems to be differences between the sexes (Bond et al., 1991). It has been suggested that this could be due to differences between sexes in hormonal control of skeletal development related to the balance of testosterone and estrogens. However, the effect of these hormones on skeletal development in the juvenile broiler is perhaps questionable since little differences between sexes in tibiotarsal length are seen until after 5 weeks of age (Classen and Riddell, 1989; Rath et al., 1996).

In addition, factors such as parent flock age and the chick's nutritional body stores at hatch may affect *e.g.* the cholecalciferol requirements of young broiler chickens (Elliot and Edwards, 1997; Vieira and Moran, 1999). Some skeletal abnormalities may be seen in the first few days after hatching, and it is possible that metabolic disorders are initiated during incubation. Skeletal mineralization starts at around 8 days of incubation, and at this time the yolk serves as a source of calcium (Johnston and Comar, 1955). There are no studies found reporting the effects of incubation environments, or breeder nutrition and management, on skeletal abnormalities in their progeny.

7. Concluding remarks

Often complex metabolic and management systems are involved in conditions of leg disorders. On many occasions, leg abnormalities due to suboptimal nutrition may be due more to the quality, consumption and absorption of feed ingredients, rather than to errors in formulation and mixing of the diets. Recommendations for certain nutrients important for optimal leg health may also need to be revised considering the relatively rapid changes in growth potential in modern broiler genotypes. Additionally, recommendations for some nutrients in relation to leg health may need to be considered in view of legislative changes within the EU regarding the prohibition of antibiotic growth promoters and certain changes in the use of raw materials. There is a continuous need to further increase the knowledge of how to optimize commercial broiler production feed for health and welfare, while maintaining production economics.

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