Metabolic Challenges and Early Bone Development

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Primary Audience: Nutritionists, Veterinarians, Live Production Personnel, Researchers

SUMMARY

Lameness is the phenotypic expression of a wide variety of genetic, conformational, nutritional, toxic, and infectious conditions. Poultry selected for rapid growth are susceptible to lameness, and it can be a significant source of mortality in heavy birds. Genetic selection has been successful in providing annual improvements in meat poultry productivity; however, some support systems, such as the cardiovascular and skeletal systems, have not kept up with the increase in body mass, making birds increasingly susceptible to compromise or failure of these systems. Breeders have been selecting for robust support systems in addition to increased productivity, but meat birds are still challenged with keeping structural and supply organs synchronized with the growth of demand tissues such as muscle. The structural system of the bird is comprised of bone, cartilage, ligaments, tendons, and the connective tissue of skin and other organs. Healthy bone growth can be disrupted by developmental, nutritional, environmental, or infectious conditions, with bone problems often being associated with more than one of these causes. A review of recent reports on bone formation and the role of trace minerals in bone development and repair indicate that availability of Zn, Cu, and Mn should be considered by nutritionists who want to enhance the resilience of bone, cartilage, and other structural tissues in their birds.

Key words: tibial dyschondroplasia, lameness, endochondral ossification, trace mineral, poultry

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DESCRIPTION OF PROBLEM

Genetic selection for efficient production has improved the performance of meat poultry, particularly broilers and turkey poults, to levels that have not been equaled by any other source of animal protein. This selection has been successful in providing annual improvements in productivity since the 1950s, with resulting increases in BW and feed efficiency that show little sign of reaching their limits. It became evident in the 1980s that some support systems, such as the cardiovascular and skeletal systems, had not kept up with the increase in body mass and that the birds were increasingly susceptible to compromise or failure of these systems associated with various deviations from stress-free conditions. Thus, any one of a variety of situations could provoke a breakdown in support systems. As commercial breeders began to select for robust support systems in addition to increased productivity, the situation has improved, but meat birds are still challenged with keeping structural and supply organs synchronized with the growth of demand tissues such as muscle. One of the most frequently affected systems is the structural bone, joints, tendons and the connective tissue of the legs and feet. This review will describe the most common structural disor-

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ders in broilers and turkey poults grown for meat yield and the various stresses that can evoke them. A review of recent reports on the role of trace minerals in bone development will serve to highlight some tactics that the nutritionist can use to enhance the ability of structural tissues to respond to stress.

BONE DISORDERS ASSOCIATED WITH RAPID GROWTH

Lameness is the phenotypic expression of a wide variety of conditions. It is common in poultry selected for rapid growth and has been estimated to affect 2 to 6% of turkey flocks [1], in which it is most common in birds older than 14 wk. Within a flock, the incidence can exceed 15% [2], and lameness is a significant source of mortality in heavy birds. It should be noted that many abnormalities in bone development can be initiated early in life, with lameness appearing only later in association with a conformational, environmental, or infectious challenge [3, 4, 5, 6]. In broilers, recent reductions in leg-related condemnations and mortalities are largely due to changes in genetic selection that, along with rigorous attention to health, nutrition, and management, have reduced the susceptibility of broilers to lameness [7]. Nevertheless, bone problems are still among the most costly disorders associated with rapid growth because of their chronicity, their tendency to reduce feed intake, and the frequency of downgrades and condemnations when affected birds are processed. Regardless of the factors provoking it, once lameness has developed, it very rarely resolves, tending to become worse with age and increasing BW. Pododermatitis, a chronic ulcerative dermatitis of the feet and hocks, contributes to progression of lameness and the performance decline associated with it.

Bone abnormalities in birds can be initiated by developmental, degenerative, nutritional, environmental, or infectious problems and are often associated with more than one of these causes. The condition advances to lameness in otherwise apparently healthy flocks that are fed high-density diets at high stocking density and that live in environmental conditions that maximize their growth rate [8, 9, 10]. Abnormal bone development can result in varus-valgus disease, tibial dyschondroplasia (**TD**), and chondrodystrophy. One cause of developmental abnormalities is imbalance – excess or deficiency – of specific hormones, including androgens, estrogens, the growth hormone-insulin-like growth factor-I system, parathyroid and thyroid hormones, and also stress-related hormones such as glucocorticoids [6]. Degenerative diseases include pododermatitis, rupture of the gastrocnemius tendon, and degenerative joint disease [8].

Nutritional stress such as vitamin or mineral deficiency or imbalance is associated with rickets and also with TD. It is important to realize that the presence of oxidized fat in the diet will cause the degradation of all fat-soluble vitamins, including vitamins D, A, and E [11]. This can reduce availability of these vitamins, sometimes resulting in lameness. It is also important to recognize that the osteolytic action of osteoclasts may be enhanced in the presence of free radicals found in poorly stabilized fat samples [12].

Among the most common infectious problems are bacterial chondronecrosis with osteomyelitis (femoral head necrosis) and tenosynovitis. Using dexamethasone injection followed by airsac challenge with *Escherichia coli* as a model for stress, Huff et al. [4] have shown that turkeys selected for fast growth are more likely to develop turkey osteomyelitis complex than birds from a slow-growing line selected for egg production.

Environmental stresses, which include high stocking density, low brooding temperatures, poor ventilation, and poor litter quality, are associated with lameness due to infected hocks, TD, pododermatitis, and tenosynovitis [8, 13]. It should also be noted that an initially minor problem such as a crack in a foot pad, if not promptly healed, can lead to lameness. An example of this is the frequency with which pododermatitis is associated with joint infections in hocks, knees, and hips [13].

It is clear that lameness is a very complex problem with many components. Genetics plays a contributing role, but lameness is closely related to the extremely rapid bone growth in broilers and turkeys fed and managed to achieve their full potential for growth [8, 9]. The structural support system has very little flexibility to respond to many commonly occurring stresses.

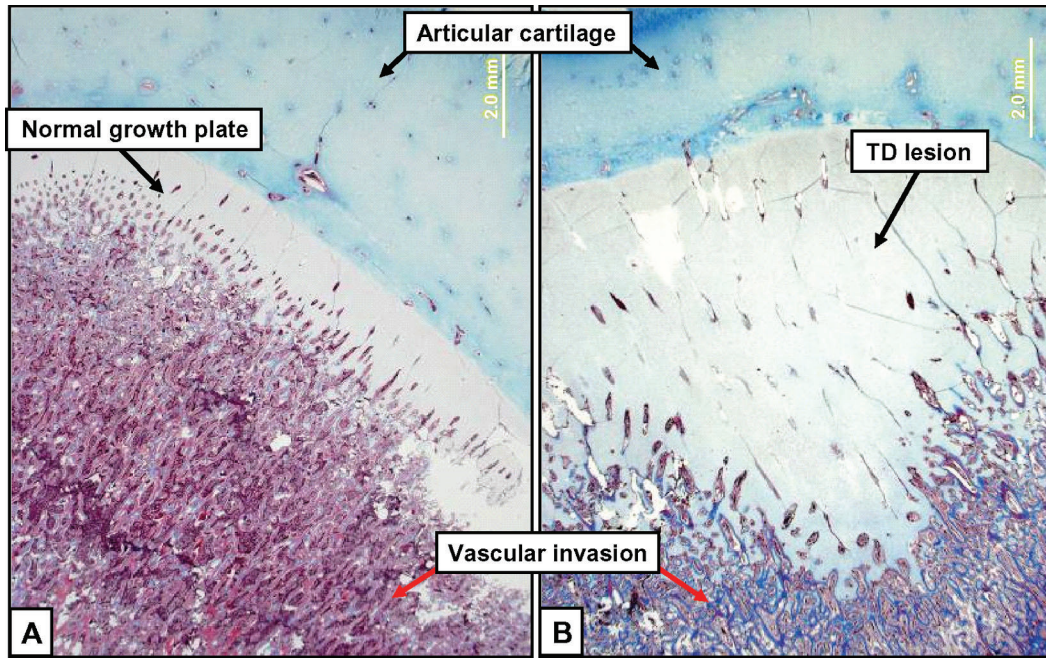

Figure 1. Histological appearance of normal (panel A) and tibial dyschondroplastic (TD; panel B) growth plates from 14-wk-old tom turkeys. Residual cartilage and lack of vascular invasion are seen in the dyschondroplastic tibia growth plate. Masson's trichrome, 10×.

TD

Tibial dyschondroplasia, the most common bone disorder in poultry, was first described by Leach and Nesheim [14]. Avian TD is characterized by an accumulation of the avascular part of the growth plate distal to the zone of proliferation. Figure 1 shows the histological appearance of a normal (panel A) and TD-affected (panel B) proximal tibia growth plate. Some authors describe TD as a failure of vascularization due to reduction in vascular penetration normally mediated by collagenolytic matrix metalloproteinases [15, 16]. There is also evidence that the defect in thiram-induced TD is related to apoptosis in endothelial cells [17]. Many studies have been done comparing the chondrocytes and extracellular matrix (**ECM**) of normal and dystrophic cartilage. The chondrocytes in TD do not fully differentiate and hypertrophy; rather, they appear to be arrested in a prehypertrophic state of differentiation [18]. The regulation of chondrocyte differentiation and gene expression is currently under study. Whether differentiation is mediated by Zn, as in other tissues, remains to be determined. Biochemically, growth plate cartilage from TD birds differs in several respects from that of normal birds [19]. Changes in osteogenic proteins such as osteocalcin and osteopontin are often accompanied by reductions in growth factors such as transforming growth factor-β and collagen type X [18].

There are conflicting data on the proteoglycan content of the ECM of TD lesions, but it has been suggested that higher molecular weight proteoglycans may inhibit vascular invasion and in this way retard ossification [18, 20]. Osteochondrosis is a developmental defect in endochondral ossification in horses and swine. The histopathology of this disorder resembles that of TD in poultry [21]. Among the nutritional conditions associated with osteochondrosis is Cu deficiency [22, 23]. In addition, Cu supplementation can prevent the TD associated with thiram administration in chicks [15, 24]. Tibial dyschondroplasia serves as an example of the leg problems that accompany rapid growth and is the most commonly studied lesion, but not all leg problems in broilers and turkeys arise from this particular pathological process. One interesting fact to note is that the progression of a

chondrocyte from the proliferative stage to the hypertrophic stage is estimated at 21 h for rapidly growing meat birds [25]. This can be compared to a 4-d period for rabbit chondrocytes [22], a 2-d period for rat, and 20-d period for human chondrocytes [26]. The selection for rapid growth in meat birds undoubtedly contributes to these differences in growth plate cellular kinetics.

BONE DEVELOPMENT

During development in ovo, a hyaline cartilage model of the appendicular skeleton is laid down [27]. The process of converting this model to bone, endochondral ossification, is begun in ovo but primarily occurs after hatch [28]. In addition, growth in the length of the long bones continues to use the endochondral ossification process by the replacement of new hyaline cartilage formed at the epiphyseal plate [29]. Many birds are marketed before epiphyseal plate closure. In this sense, the axial bones of most meat poultry never reach the stage of homeostasis and remodeling characteristic of adult vertebrates but are undergoing development throughout the life of the bird [30]. Indeed, Rath et al. [31] observed that the longitudinal bone growth of broiler breeders continued until 25 wk of age. This is important, because the processes of bone dissolution and reformation are coupled in bone remodeling but uncoupled during initial bone deposition [27].

Endochondral Ossification

The process of endochondral ossification after hatch involves a highly coordinated sequence of events that begins with proliferation of cartilage cells (chondrocytes) in the growth plate. The spatial coordination of this process is reflected in the microscopic anatomy of the epiphyseal plate. At the proximal edge of the plate are resting chondrocytes, below which are the proliferating chondrocytes responsible for new growth. As these cells mature, they secrete the ground substance of hyaline cartilage, including large aggregating sulfated proteoglycans such as aggrecan [32], and the fibers in which they are embedded, primarily type II collagen [33]. The chondrocytes encase themselves in this avascular ECM, through which all nutrients and waste products must diffuse. There are many other components in the ECM, including growth factors, cytokines, prostaglandins, and small molecular weight proteoglycans. The function of many of these smaller molecules is still being described [15, 34, 35, 36]. As the chondrocytes age, their secretory product changes, as does their morphology. The distal end of the growth plate is called the hypertrophic zone, where the chondrocytes enlarge and undergo terminal differentiation [37, 38]. At this time, their secretory products change to include collagen type X as well as type II and other proteins such as annexin and fibronectin [20]. The proteoglycan components are smaller molecular weight molecules such as decorin and byglycan [19, 39]. This zone is also characterized by its high level of alkaline phosphatase activity, which is associated with mineralization. Chondrocytes in the distal hypertrophic zone (the zone of provisional calcification) also synthesize collagen type I, in common with the invading bone precursor cells, osteoblasts [20].

The molecular and cell biology of endochondral ossification are still being described [40]. Mineralization, in particular, is restricted to a small portion of the cartilaginous bone model, the epiphyseal plates, and is controlled both spatially and temporally. Either insufficient or excessive mineralization is associated with significant pathology such as osteoporosis and osteopetrosis. Briefly, as the chondrocytes age and exhaust their supply of glycogen, their mitochondria begin to concentrate Ca [20]. This occurs in conjunction with a shift to anaerobic metabolism accompanied by changes in O_2 tension and glycolytic enzymes [33]. The hypertrophic cells begin to shed matrix vesicles, membrane-bound microstructures averaging 30 to 100 nm in diameter [41, 42]. These vesicles contain several specific proteins, including annexins II, V, and VI; alkaline phosphatase; and a core complex of Ca and P that forms before shedding [20]. The complex also includes phosphatidyl serine as a component [43, 44]. These amorphous Ca-P complexes appear to serve as the nucleation site for calcification in endochondral ossification. The annexin mediates Ca^{2+} influx into the vesicles and also binds directly to types II and X collagen, anchoring the vesicles to the ECM [45]. The matrix vesicle

also includes Zn and Cu, and these are thought to have a role in maintaining the amorphous structure of the mineral until the initiation of hydroxyapatite crystallization [46]. Interestingly, a histochemical study of the distribution of Zn in bone tissue detected the mineral at the borderline between calcified and uncalcified material. Zinc staining was associated with the late-stage hypertrophic chondrocytes near the front of vascular invasion, which is consistent with a role for Zn in regulating matrix vesicle mineral crystallization as described above [47]. The membrane and lipid components of the matrix vesicle are not representative of the cell membrane of the hypertrophic chondrocyte [48]. In particular, the matrix vesicle membrane is enriched for phosphatidylserine [41]. The crystallization of hydroxyapatite is initiated in the matrix vesicles. The apatite crystals rupture the vesicle membrane and begin directed growth along the type I collagen fibers synthesized by hypertrophic chondrocytes [20]. Research has suggested a role for type I collagen in hydroxyapatite crystal elongation, orientation, and propagation [49, 50, 51, 52].

As these initial mineralization steps are occurring at the distal edge of the growth plate, invasion of the cartilage model by the vascular system simultaneously delivers bone precursor cells (osteoblasts) to the interior. This initiates the actual process of bone formation [27]. There is evidence that chondrocytes at the chondroosseous junction express angiogenic factors such as vascular endothelial growth factor [53] and transferrin [22] and in this way stimulate the invasion of blood vessels into the model. Degeneration of the cartilage matrix is mediated by collagenase-3, a metalloproteinase containing Zn in the active site [22]. The vessels bring the osteoblasts, which bind to the mineralized cartilage matrix and begin to secrete the components of the bone extracellular matrix, osteoid. The collagen of osteoid is type I. The crosslinking of this collagen provides the structure along which hydroxyapatite crystals grow [45].

These events make it clear that proper bone formation is a highly complex, multistep process that depends on much more than Ca and P. Multiple cell types must act sequentially and in concert for proper development. Each step is governed by transcription factors and signaling polypeptides [54]. It is also important to recognize that this initial ossification does not yield bones that are structurally mature and mechanically robust. Following initial deposition of ECM and mineral, important changes occur that are responsible for the increase in bone breaking strength observed in older birds [55]. For example, although mineral deposition and crystal maturation is essential for compressive strength, the alignment and crosslinking of the collagen in the ECM is highly correlated with bone breaking strength. One interesting thing about bone is that all of these critical posttranslational maturation steps take place outside the cells, within the ECM.

Trace Minerals in Bone Development

Although trace minerals are not often considered in the context of bone development, endochondral ossification absolutely depends on trace mineral availability. Manganese is essential for the formation of the mucopolysaccharides that form the ground substance of the cartilage model [27]. Copper is in the amorphous mineral of the matrix vesicles, preventing its premature crystallization [56], and also plays an important role in the crosslinking of collagen and elastin, which gives bone its tensile strength and elasticity [57]. Zinc plays an important role in regulation of hydroxyapatite crystallization [56, 58], collagen synthesis [59], and the cellular invasion of the cartilage matrix by the osteoblasts. This invasion requires the activity of the collagenase-matrix metalloproteinase molecules, particularly collagenase-3, which contain Zn in their active sites [22, 60]. The fact that intracellular Zn fluxes are associated with apoptosis in growth plate chondrocytes [61] suggests that Zn may play a role in endochondral ossification beyond its participation in vascular invasion and hydroxyapatite crystallization [62, 63, 64]. Zinc is also associated with the changes in gene transcription that accompany ossification. Furthermore, recent work indicates that the deficiency of a specific Zn finger protein (the Gli2 transcription factor) is associated with impaired endochondral ossification in mice [65]. In this study, the delayed ossification was associated with aberrant gene expression, including decreased expression of a variety of angiogenic factors in hypertrophic chondrocytes.

Our group has been studying the role of trace mineral availability on bone development and abnormalities associated with rapid growth. The importance of Ca, P, and vitamin D has been so well documented that it is rare to encounter bone abnormalities associated with simple deficiencies in any of these components [66]. What is not as well understood is that the very process of ensuring adequate Ca and P may cause a deficiency in trace minerals. This is due to the tendency of cationic minerals to form insoluble complexes with free P, phytic acid, and other components of the digesta. As more Ca is fed, less Zn, Cu, and Mn are absorbed [66]. This is partly due to the coordinate covalent binding of all of these cations to phytic acid. Phytate is able to form chelates of these minerals that are very stable and highly insoluble [67]. The antagonism is mutual in that the binding of Ca, Zn, and other minerals also reduces the availability of phytin P, even in the presence of exogenous phytase [68]. Antagonisms also occur between one trace mineral and another. For example, high levels of Zn reduce the availability of Cu [69]. The absorbability of Mn, generally low under many circumstances, can be reduced in the presence of supplemental P [70]. Availability of inorganic sources of minerals can also be reduced by other nutrients; for example, Cu availability is reduced by ascorbic acid [71].

Historically, trace minerals have been supplemented in poultry diets using the inorganic salts such as Zn, Cu, and Mn oxide or Zn, Mn, and Cu sulfate. Inorganic minerals have also been used in the development of mineral requirements for poultry diets [72]. However, because of the antagonisms described above, use of inorganic salts can result in variable or low bioavailability of the mineral.

The common denominator in the antagonistic interactions among minerals is the dissociation of the inorganic salt in the relatively low pH of the upper gastrointestinal system. When the mineral reaches the higher pH of more distal gut segments, it can bind to many minerals, nutrients, and nonnutritive components of the digesta, such as phytate and fiber, which render it insoluble. Insoluble forms of minerals are excreted.

The advantage of organic trace minerals is that the binding of the mineral to the organic ligand provides stability of the complex in the upper gastrointestinal system. Organic trace minerals should resist dissociation in the crop, proventriculus, and gizzard, thus allowing the intact complex to be delivered to the absorptive epithelium of the small intestine [73]. The organic forms of many minerals, including Zn, Cu, and Mn, are widely used in animal agriculture. Increased availability of organic Zn and Cu compared to inorganic forms has been demonstrated [74, 75, 76]. Increasing the availability of trace minerals can have significant biological effects, including improved bone mineralization [77, 78, 79]; however, important environmental benefits also accrue from the reduced excretion of those minerals into the environment [80, 81].

Availability of trace minerals, particularly the combination of Zn, Cu, and Mn, plays a critical role in early development because of the integrated function of metalloenzymes in building structural connective tissue. Although collagen synthesis requires Zn, unless sufficient Cu is present, fibrils will not be properly cross-linked, and the resulting structure may be weakened or may fail [31]. The development of appropriate connective tissue requires that compliant organs such as the gut be provided with the capability to accommodate changes in digesta volume at the same time that structural tissues such as tendon be highly resistant to stretching in response to a change in mechanical tension. The ECM of bone contributes tensile strength, whereas the crystalline mineral provides rigidity and compressive strength [31, 39]. Both are essential for bone to be a flexible, strong, and lightweight structural material.

The ground substance of tissue, particularly the proteoglycan matrix in which collagen and elastin are embedded, requires Mn for glycosylation of the protein core molecule [66]. Proteoglycans vary in their hydration and aggregation, and these differences are essential to their various structural roles. For example, the highly hydrated proteoglycans of the articular surface of joints provide a cushion to protect the underlying bone [82]. In this example, the expansion of the massive proteoglycans is limited by a collagen network of very high tensile strength that compresses without collapsing. Bone, on the other hand, resists compression even though the organic components of its connective tissue are

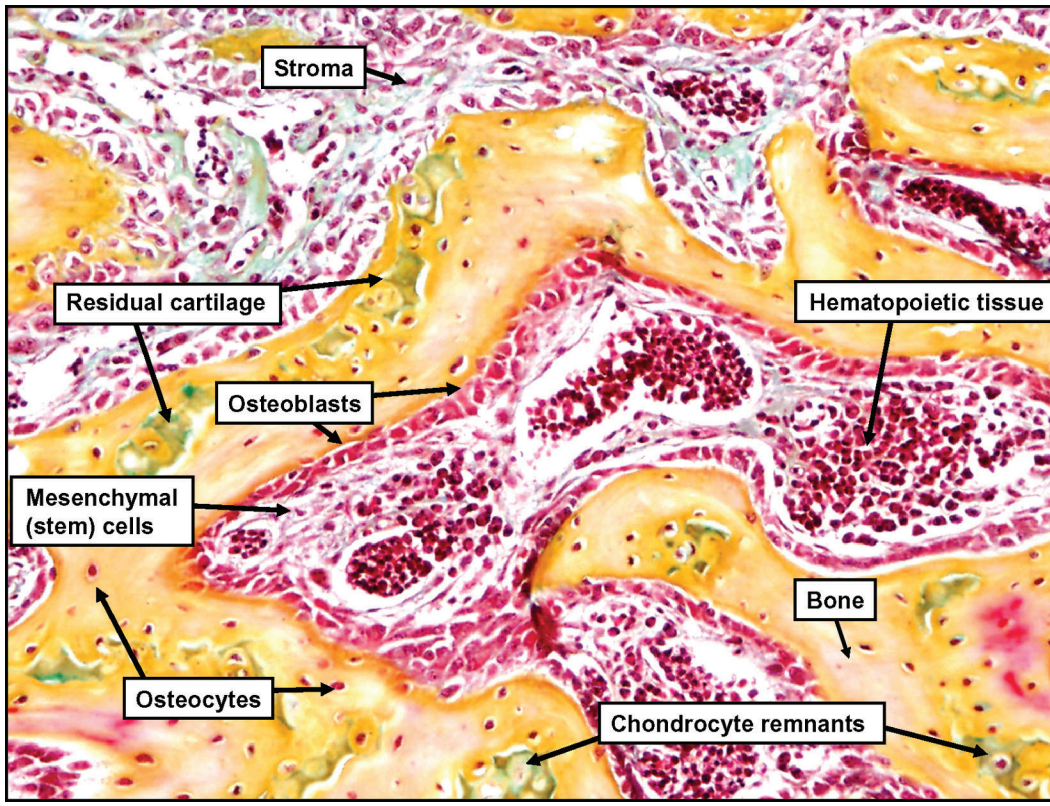

Figure 2. Micrograph illustrating cellular relationships in bone marrow of 14-wk-old tom turkeys. New bone (yellow) and the residual cartilage (green) within it create a niche for hematopoietic cell proliferation and differentiation. Mature bone cells (osteocytes) can be seen encased in mineralized matrix, whereas lacunae with remnants of chondrocytes are still visible at the center of bone trabeculae. Cuboidal osteoblasts line the endosteal surface and lay down osteoid for mineralization. Movat's pentachrome, 300×.

similar to that of cartilage. It is important to note, however, that the collagen present in the articular cartilage is type II, a network-forming collagen, whereas that of bone is predominantly type I, a fibril-forming collagen [22]. Thus, the large hydrated proteoglycan (aggrecan) is constrained by the collagen network and releases water in response to compressive force. The type I collagen of bone, however, forms large, cross-linked fibers aligned in response to tensile force and in this way reinforces the strength of the hydroxyapatite mineral crystals [82].

NUTRITIONAL INTERVENTION AGAINST LEG PROBLEMS

Thousands of studies of the effects of specific nutrients on leg problems in poultry have been reported over the last 40 yr. This body of research, recently reviewed by Whitehead [83] and Oviedo-Rondon and Ferket [84], highlights the importance of the macrominerals Ca and P and their relative ratio on bone development. It has been clearly shown that vitamins D, A, C, K, and most B-vitamins are essential for optimal bone development. High levels of the S amino acids, Met and Cys, as well as the S-containing metabolite homocysteine, particularly associated with marginal vitamin B_6 deficiency, are associated with lameness due to abnormal bone collagen [85].

An interesting body of work has demonstrated the effect of fatty acid nutrition on bone mineralization and mechanical properties [86, 87, 88]. Providing fats containing relatively higher levels of n-3 fatty acids to laying hens is associated with increased total n-3 fatty acids in progeny yolk lipid and reduction in progeny tibial prostaglandin E_2 production. These changes

are associated with increasing tibial shear force, percentage of bone ash, bone collagen pyridinium crosslinks, and diaphyseal cortical thickness in progeny bones [87]. These results add to a growing body of data linking inflammatory cytokines and other immune mediators with bone cell differentiation, both anabolic (chondrocytes and osteoblasts) and catabolic (osteoclasts) [54, 89]. For example, an acute inflammatory response initiated by *E. coli* lipopolysaccharide can dramatically affect bone metabolism and homeostasis, resulting in decreased tibia Ca and tibia breaking strength [90]. This is perhaps one example of the complex cellular relationships within the bone marrow, i.e., between the hematopoietic stem cells and bone marrow stromal cells of the endosteal surfaces [91], which are providing new insights into the molecular biology governing the relationship between the myeloid and osteogenic lineages [92]. Indeed, the pluripotent stem cell ($CD34^+$ hematopoietic bone marrow cell) of red blood cells, immune cells, and osteoclasts resides near the mesenchymal precursors of bone cells (osteoblasts) [93]. In addition, osteoblasts have been shown to secrete factors that affect the development of hematopoietic stem cells – factors such as granulocyte colony-stimulating factor, macrophage colony-stimulating factor, and interleukin-1 and interleukin-6 [93]. It would be interesting to test whether genetic selection for increased performance has exerted an effect on the common stem cell of the lymphoid and myeloid lineages to result in altered lymphoid, macrophage, and osteoclast differentiation potential [27]. This could yield the tendency to low adaptive immunity, high innate immunity, and poor bone stability phenotype seen in modern meat birds.

Figure 2 is a micrograph from the tibia of a 14-wk-old turkey. It shows the intimate physical proximity of bone and hematopoietic tissue. Although they reside together and act in coordinated opposition in the bone, the anabolic and catabolic cells of the bone have different origins, one related more to connective tissue and adipose (chondrocytes and osteoblasts), and the other is more related to blood and immune cells (osteoclasts) [27]. The transcription factors and signaling polypeptides of these cells and their precursors are currently being described, and this research is yielding fascinating details about the regulation of bone and immune function. For example, some signaling molecules act in the differentiation of bone and immune cells, including osteoprotegrin, interferon-γ, interleukin-6, tumor necrosis factor-α, and receptor activator of nuclear factor-κB ligand [54, 91, 93, 94]. These have begun to reveal the tightly regulated and highly coordinated interactions between the 2 tissues. Such studies may well provide new avenues to approach bone development and the effect of inflammation and infection on bone homeostasis.

An example of the interaction between immune challenge and bone homeostasis was observed recently in studies of the effect of an organic source of Zn on the response of birds to coccidiosis vaccination and challenge [95]. In these studies, the organic trace mineral source was Mintrex Zn [96] organic trace mineral, which is a chelated Zn source using 2-hydroxy-4-methylthiobutanoic acid as the organic ligand. In studies comparing this product to inorganic sources of Zn, improvements in tibia Zn deposition over that seen with Zn sulfate were observed in broilers, and the improvement in whole body Zn status was most striking following a coccidiosis challenge [95]. A recent publication by Predieri et al. [97] confirmed the increased availability of Zn from a chelate made with 2-hydroxy-4-methylthiobutanoic acid resulted in a reduction in Zn excretion from the animal.

A recent field trial by Richards et al. [98] tested a blend of organic Zn, Cu, and Mn (Mintrex P organic trace mineral [96]) for the ability to improve leg and foot problems in heavy turkeys. These turkeys were exhibiting lameness that involved pododermatitis, TD, synovitis, and varus-valgus disease. The incidence was typical of that seen in commercial turkeys, with relatively minor foot and leg problems seen early and progressing to overt lameness at 14 wk and above. The treatments in this study consisted of the existing dietary regime and the same diet containing the organic trace mineral blend. The existing regime contained supplemental inorganic trace minerals and 25-hydroxycholecalciferol. Birds at ages 0 to 6 wk, 6 to 12 wk, 12 to 16 wk, or 16 to 20 wk were necropsied by selecting 3 lame and 2 normal birds from each of 8 flocks per month for 7 mo. Age and weight of individual birds as well as incidence of TD,

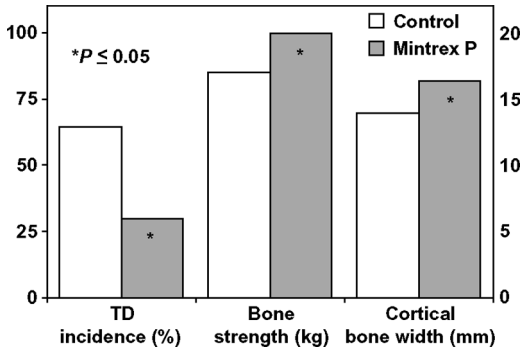

Figure 3. Effect of an organic trace mineral blend (Mintrex P [96]) added to a diet containing inorganic trace minerals on incidence of tibial dyschondroplasia (TD; left axis), tibia breaking strength (left axis) and cortical bone width (right axis) in 30- to 40-lb tom turkeys. Reduction in TD and increased cortical bone deposition may have contributed to the significant increase in bone breaking strength seen with the organic trace mineral.

rickets, synovitis, and foot pad lesions were recorded. Tibial bones were measured for length, diameter, cortex thickness, bone breaking strength, and ash. Incidence of TD, synovitis, and foot pad lesions increased with age, as did foot pad scores, bone strength, and ash content. Lameness was positively correlated with synovitis ($P < 0.05$). Bone breaking strength was significantly correlated with width of cortical bone ($P < 0.0001$). As illustrated in Figure 3, the feeding of this organic source of trace minerals significantly ($P < 0.05$) reduced TD incidence and increased both bone breaking strength and cortical bone thickness in the heaviest birds (>30 lb). In addition, the organic mineral treatment was also associated with a significant reduction in incidence of synovitis, incidence of foot pad lesions, and also foot pad lesion severity (data not shown). Clearly, in this case, despite the provision of Ca and P, and in the presence of 25-hydroxycholecalciferol, increasing trace mineral bioavailability was able to significantly improve the quality of structural tissues in the heaviest birds.

CONCLUSIONS AND APPLICATIONS

1. Bone development in rapidly growing birds is easily disrupted by deviations in nutrient availability and environmental quality. The structual system in poultry selected for meat yield is only marginally capable of matching the growth rate of the muscle. Thus, management and nutrition must constantly be examined and improved as needed.
2. Abnormalities in development and metabolic challenges early in life can be manifested later in lameness and poor performance. Once initiated, problems in bone ossification can lead to weakness in legs or in hock, knee, or hip joints that culminates in immobility or condemnation.
3. There is no one cause of lameness in these birds; the problem is multifactorial. Nevertheless, changes in growth rate, management, or nutrient availability can improve structural performance, resulting in decreases in factors associated with lameness such as TD.
4. Nutritionists need to consider trace mineral availability, particularly when feeding high levels of Ca and P. Mineral antagonisms can cause a secondary deficiency in Zn, Cu, or Mn, all of which are required for normal bone development and remodeling.

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