

PRINCIPLES OF BONE DEVELOPMENT IN HORSES

LARRY A. LAWRENCE

Kentucky Equine Research, Versailles, Kentucky, USA

Few animals are as precocious as the horse. Within 20 minutes of birth a foal may stand, and within hours can be ready to run at speeds no human athlete will ever achieve. At this stage of life, even with this exceptionally early development, horses have only 17% of their mature bone mineral content, but they also have only 10% of their ultimate body weight. The relationships between growth, nutrition, bone strength and development, body weight, and the forces applied to bone are all orchestrated in a careful balance when optimal growth is achieved.

The selection and breeding of horses for desirable traits have been practiced for over 2000 years. However, most of what we have learned about the growth of horses has been recorded in the past 20-30 years. In 1979 Dr. Harold Hintz reported Windfields Farm's growth data for 1,992 foals from birth to 22 months of age. The records illustrate how quickly foals grow. Thoroughbreds and other light horse breeds will reach 84% of their mature height at six months of age. Assuming a mature Thoroughbred will be 16 hands, the six-month-old weanling will be approximately 13.2 hands. At 12 months that horse will have reached 94% of its adult height or around 15 hands, and at 22 months it has almost finished growing in height, reaching 97% of its full height at approximately 15.2 hands. Mature weight is reached at a slower rate; during the first six months of life, the foal will gain 46% of its mature weight. Assuming a mature weight of 500 kg, the six-month-old will weigh approximately 230 kg. At 12 months it will have reached 65% of its mature weight (325 kg), and at 22 months it should be 90% of its adult weight (450 kg). Average daily gains described by Hintz are the same as those recommended by the National Research Council (NRC) for moderate growth. The NRC reports that six-month-old weanlings with a projected adult weight of 500 kg gained 0.65 kg per day. Twelve-month-old yearlings gained 0.5 kg per day, and 18-month-old long yearlings gained 0.35 kg per day (Table 1).

Radiographic studies on the acquisition of bone mineral in horses from one day of age to 27 years have shown that maximum bone mineral content (BMC) is not achieved until the horse is six years old. If the rate of mineralization of the cannon bone and age are compared, a pattern emerges that is more similar to that of weight gain than height. At six months of age horses have attained 68.5% of the mineral content of an adult horse, and by one year of age they have reached

76% of maximal BMC. Bone is a much more dynamic tissue than it appears to be upon casual observation; however, complete bone mineralization lags behind growth in height and weight.

Table 1. Projected growth parameters for a young horse.

<i>Age</i>	<i>Height</i>	<i>% Mature Height*</i>	<i>Weight</i>	<i>% Mature Weight*</i>
6 months	13.2 h	84%	230 kg	46%
12 months	15.0 h	94%	325 kg	65%
22 months	15.2 h	97%	450 kg	90%

*Estimates based on 16-hand, 500-kg mature horse.

A basic understanding of the process of bone development helps to explain the complicated nature of growth in the horse. There are two anatomical types of bones in the skeleton. Flat bones are generally for protection and include the skull, mandible, and ilium. Long bones are found in the appendicular skeleton and include the metacarpal, humerus, and femur. These bone are different from a functional perspective, and they are developed by two distinctly different processes. The flat bones are developed by intramembranous ossification and the long bones are developed by endochondral ossification. Long bone development is generally of greatest interest because of its impact on the soundness of the horse.

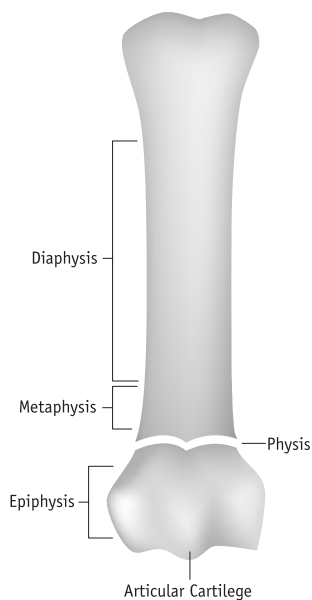


Figure 1. External examination of the third metacarpal.

External examination of the third metacarpal (Figure 1) shows wide extremities at the ends of the bone (epiphyses), a cylindrical tube tapering to a central waist in the middle (midshaft or diaphysis), and a developmental zone (the metaphysis and physis) between them. In the growing long bone, the physis or growth plate is a layer of proliferating cells and expanding cartilage matrix. The growth plate is calcified, remodeled, and replaced by bone at the end of bone growth. On cross-section the bone marrow cavity is a central cylindrical tube. The waist of long bones contains the most densely calcified area of cortical bone. Looking from the waist to the metaphysis and epiphysis, the cortex becomes thinner and the internal space is filled with a network of thin, calcified trabeculae known as cancellous bone or trabecular bone.

There are two surfaces at which the bone is in contact with soft tissue: an external surface (periosteum) and an internal surface (endosteum). These surfaces are lined with layers of osteogenic cells. The endosteum is more active metabolically because of the structural differences in compact and trabecular bone. Between 80 and 90% of the volume of compact bone is calcified, whereas only 15-20% of the trabecular bone is calcified. Trabecular bone is in close contact with bone marrow, blood vessels, and connective tissue. The endosteal bone surface is 70% of the interface with soft tissues (Baron, 1999). The strength of compact bone helps it fulfill its mostly mechanical function, and trabecular bone is more labile and metabolically active.

Bone is formed by collagen fibers usually oriented in a preferential direction and noncollagenous proteins. Spindle- or plate-shaped crystals of hydroxyapatite $[3\text{Ca}_3(\text{PO}_4)_2](\text{OH}_2)$ are found on the collagen fibers within them and in the ground substance. They tend to be oriented in the same direction as the collagen fibers. The orientation of the fibers alternates in mature bone from layer to layer, giving the bone a lamellar structure. When bone is formed rapidly during development, collagen fibers are loosely packed and randomly oriented.

Endochondral ossification occurs in an embryonic cartilage model. Longitudinal growth occurs at the growth plate in a series of zones. The first zone contains resting chondrocytes. The second zone is where chondrocytes divide and synthesize matrix; it is called the proliferative zone. The hypertrophic zone is the site where maturing chondrocytes become larger and produce alkaline phosphate and calcium matrix vesicles. The fourth zone is the calcified zone. The process of mineralization begins when capillaries and osteogenic cells from the diaphysis invade the columns of chondrocytes. The chondrocytes in the newly calcified zone die. Osteogenic cells multiply and differentiate into osteoblasts. Osteoblasts arrange themselves along the remnants of the cartilaginous trabeculae and produce the matrix constituent's collagen and ground substance. Membrane-bound cellular bodies released from chondrocytes and osteoblasts facilitate mineralization. These are known as ECM vesicles and they contain calcium and phosphorus. In addition, they provide enzymes that can degrade inhibitors of mineralization. Alkaline phosphatase hydrolyzes phosphate esters, increasing phosphatase concentration,

which in turn increases mineralization. Osteoblasts are transformed into osteocytes or bone cells in calcified matrix. Osteocytes are found embedded deep within bone in osteocytic lacunae. A network of thin canaliculi permeating the entire bone matrix connects osteocytes.

The bone cells associated with resorption of bone are osteoclasts, giant multinucleated cells found in contact with a calcified bone surface lacuna. Osteoclasts resorb bone via lysosomal enzymes (e.g., acid phosphatase and cathepsin K) and collagenase. The enzymes are secreted through a ruffled border. Resorption starts with digestion of the calcium and phosphorus-containing hydroxyapatite crystals. Then the collagen fibers of the matrix are digested by collagenase or by action of cathepsins at low pH.

The process of long bone growth involves the development of bone tissue and the resorption of bone tissue at the same time. As bone develops at the growth plate, it forms the metaphysis. The metaphysis is flared outward with a narrow cortex. Growth must take place within the context of the structural and metabolic functions of the bone. This is accomplished by continual appositional growth on the endosteal side of the bone and active removal of bone by osteoclasts on the periosteal surface.

This brief description of bone development should help clarify the complicated and metabolically sensitive nature of the process. Optimal growth rates may vary somewhat between breeds, but all young horses have several critical considerations for bone growth and development. Extremely rapid growth caused by overfeeding (particularly energy) has been implicated in developmental orthopedic disease (DOD) and unsoundness. Periods of slow or decreased growth followed by rapid growth are particularly dangerous. Imbalanced levels of calcium, phosphorus, and trace minerals have been linked to DOD. Certain types of forced exercise also seem to cause bone development problems.

Bone development begins before birth and continues beyond 18 months of age. The period between three and nine months of age appears to be the most precarious for the foal in terms of DOD. During this time, serious conditions can develop that might restrict the athletic potential of a horse. It is important to monitor growth rates and evaluate the foal's skeletal development. Steady, moderate growth along a typical growth curve appears to provide the best method of reducing developmental problems.

Kentucky Equine Research has been weighing and measuring foals, weanlings, and yearlings monthly in central Kentucky for over 10 years. Those records combined with numbers from universities and Windfields Farm in Canada have resulted in a tremendous vault of comparative growth data. These data have been formulated into software designed to track growth and make comparisons with databases containing records of thousands of foals. Observations from research trials and practical experience have led to the recognition that slow, steady growth is best for horses. Pagan (1998) reported on the incidence of developmental orthopedic disease (DOD) in Thoroughbred foals. A total of 271 foals was

monitored. Ten percent of the foals were diagnosed with DOD. Osteochondritis dissecans (OCD) lesions of the fetlock were diagnosed at an average age of 102 days in fillies (2% affected) that were small at 15 days of age (3.18 kg below average). Stifle and shoulder OCD lesions were diagnosed at the average age of 336 days. About 2% of the foals were affected, and they tended to be 5.45 kg above the average weight at 25 days and 31 pounds above average at 120 days. Foals that developed hock OCD lesions tended to be heavier than average at birth and were 8.18 kg above average at 15 days. They had higher average daily gains until 240 days, and they were 15.45 kg above average at that age. Pagan recommends that managers (1) record birth weights; (2) express weights as a percent of a reference; (3) do not allow deviation of 15% or more from the reference weights; (4) keep records of DOD and management changes; (5) weigh monthly; (6) do not overfeed lactating mares; (7) provide adequate turnout; and (8) consider weaning early if foals are growing too fast.

All efforts to support continuous steady growth in foals are important in helping to limit bone development problems. Foals that have had slowed growth followed by large growth spurts are at particular risk for DOD.

References

- Baron, R. 1999. Anatomy and ultrastructure of bone. In: J.B. Lain and S.R. Goldring (Eds.) *Primer on the Metabolic Diseases and Disorders of Mineral Metabolism*. (4th Ed.) p. 3-10. Lippincott, Williams & Wilkins, Philadelphia, PA.
- Hintz, H.F., R.L. Hintz, and L.D. van Vleck. 1979. Growth rate of Thoroughbreds: Effect of age of dam, year and month of birth, sex of foal. *J. Anim. Sci.* 48:480.
- Pagan, J.D. 1998. The incidence of developmental orthopedic disease (DOD) on a Kentucky Thoroughbred farm. In: J.D. Pagan (Ed.) *Advances in Equine Nutrition*. p. 469-475. Nottingham University Press, Nottingham, U.K.