As you admire a horse, you take in his physique, his muscle tone, and the sheen of his coat. But underlying these essential outer parts is the structure that forms the scaffold to which all soft tissues attach: the equine skeleton.

The skeletal system is made of many interconnected tissues: bone, cartilage, tendons, and ligaments. The length of the bones and the angles at which they come together dictate a horse’s conformation, way of going, stride length, and potential power as an athlete.

Skeletal Parts

The components of the skeleton are categorized as: a) the axial skeleton (skull, vertebral column, ribs, and sternum), and b) the appendicular skeleton (forelimbs and hind limbs).

The horse’s skeleton is a remarkable system of various bone types, capable of remodeling and adapting in response to exercise and training.

Equine Skeletal System
The vertebral column, ribs, and sternum stabilize the torso and facilitate flexibility for rapid changes in direction. Long bones connect to each other at joints, which allow limb flexion. This joint construction shortens the lever arm, decreasing the force and energy required for limb movement. Muscles and tendons (see page 23) retract or protract a leg, changing the horse's center of gravity as he moves, and the entire skeletal system makes continual adjustments in accordance with limb placement.

Each of the 205 equine skeletal bones provides anchor points for ligaments that stabilize bone connections at joints, and for muscle-tendon units to achieve efficient contractions to move the limbs.

The late James Rooney, DVM, in his book, *The Lame Horse*, painted an image of how the muscle-tendon unit works: Hang a string with weights on each end and jerk the string attached to the weights—the weights will move irregularly. But, if you tie an elastic cord (representative of a tendon-muscle unit) between the weight and a solid object, jerking the weight initiates a smooth pendulum swing with minimal vibration. This dampening effect is similar to what happens as muscle-tendon units initiate movement of the skeleton.

**Skeletal Evolution**

The tiny, three-toed prehistoric horse, *Eohippus*, grew to today's modern stature through elongation of the bones of the appendicular skeleton. Consider your hand and wrist—your wrist bones equate to a horse's carpus (knee), your third knuckle (the one closest to your wrist) is his fetlock, and his cannon bone, pastern bones, and coffin bone are equivalent to the phalangeal bones of your middle finger.

The other digits disappeared, and the only remaining vestiges are visible as tiny splint bones.

A horse's stifle is similar to your knee, his hock equates to your ankle, and the other distal limb bones are telescoped in length and correspond to the metatarsals and phalanges of your foot (but, once again, the phalangeal bones are equivalent to those in the middle digit of your foot).

Equine skeletal components have evolved over the years to allow the horse to run faster—a survival characteristic required for fleeing from predators. As the limb bones of the appendicular skeleton elongated, muscle-tendon units moved upward closer to the center of mass in the thorax. This improved propulsion and acceleration, making a horse fleet of foot. In addition, bone mass of the lower (distal) limb portions has been pared down, reducing the energy required for locomotion.

To complement this more energy-efficient skeletal system, flexor tendons store elastic energy that can be released to move the bony lever arms quickly in a ground-covering stride. For example, consider the multiple muscle bodies (triceps) that attach to the olecranon (point of the elbow). The relatively short lever of these muscles moves the long lower forelimb in large arcs to achieve stride length.

Besides acting as lever arms to elicit locomotion, the different skeletal tissues absorb impact concussion to varying degrees and provide a mineral reservoir of calcium and phosphorus that helps the horse's body maintain important metabolic functions.

**Growth and Development**

While they're only about a millimeter wide, growth plates (physes) in a young,
Equine Skeletal System

growing horse enable impressive longitudinal growth of the long bones, giving a horse his height and forming his structural conformation. At a certain age, which differs for various joints, cartilage within the growth plates is replaced with bone tissue; then long bones cease to elongate further.

The tissue between joint cartilage and the body of the bone that provides structural support for adjacent cartilage is referred to as subchondral bone. This is where developmental orthopedic disease (DOD) problems such as osteochondrosis arise, in which cartilage does not fully develop and, instead, forms a cyst or flap.

Normal functional adaptation, particularly in response to exercise, stimulates the growth plates and also enables subchondral bone to withstand limb loading and athletic challenge. Growing horses that are exercised have a reduced incidence of osteochondrosis defects compared to those with limited exercise.

The ability of skeletal tissue to respond to mechanical stimulation provides an invaluable tool to “shape” a durable athlete. For example, optimal changes in joints important to future strength and resistance to injury occur before a foal is 5 months old.

It’s important to take advantage of a window of opportunity in a horse’s early life when you can provide regular turnout and exercise to ensure future skeletal resilience and durability. Researchers have focused on the impact of confinement on skeletal development of young horses, with the unequivocal conclusion that restriction of exercise in a young, growing foal retards cartilage and bone development. However, if a foal is eventually provided with pasture exercise, these tissues might develop within normal limits.

Conversely, introducing too much exercise to a growing support system might be counterproductive; cartilage injuries that cannot heal might be a prelude to osteoarthritis development. Allowing young horses to play at pasture achieves sub-maximal loading of the skeletal system, whereas confining them to a box stall creates conditions for injury, particularly if short bouts of heavy exercise are superimposed on unconditioned joints, tendons, and bone.

Moderate exercise “trains” skeletal tissues to accept weight-bearing and limb-loading forces within reasonable limits. A recipe for forced exercise is impossible to determine; some forced exercise might be too rigorous and potentially injurious.

During the growing years, excess activity can lead to early fusion of growth plates and/or angular limb deformities, thereby limiting a horse’s potential size or affecting his conformation. Any injury that reduces limb loading might retard limb elongation.

Nutrition and genetic factors play important roles in determining even growth of the appendicular skeleton. Musculoskeletal growth occurs not as a steady continuum, but rather as a series of differential growth “spurts” more like a ratcheted pattern than a smooth curve. Rapid growth phases present feeding challenges—excessive nutrition (even more so than inadequate nutrition) is a common culprit in the history of young horses that develop DOD. Regular reassessment of the diet, along with micromineral (copper, zinc, calcium, phosphorus) and forage analysis, ensures that you’re offering the animal an appropriate diet.

Conformational characteristics influence mechanical loading of the skeletal system. Therefore, uneven weight distribution down the bony column (often evidenced by abnormal hoof wear) also place uneven stresses on growth plates.

O.R. Adams, DVM (author of Lammess in Horses), noted that “faulty conformation is not an unsoundness, but rather is a warning sign.”
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Skeletal Adaptation to Exercise

William Jones, DVM, in *Equine Sports Medicine*, reported that most necessary information is coded genetically such that a recognizable bone will form even in the absence of functional activity. Structural competence of bone depends on the total amount of bone tissue present, its girth, cortical thickness, cross-sectional shape, and density and arrangement of its internal architecture. Each of these features is influenced by functional activity, yet it can be modified by training.

Functional adaptation of skeletal tissues is useful in developing a talented equine athlete for recreational pursuits. Bones become progressively stronger when owners and trainers strategically apply controlled training regimens in gradually increasing increments.

The adaptive response of bone to loading stress is called “remodeling.” Exercise has a training effect on the skeleton by adjusting the mass and arrangement of bone tissue without modifying the nature of the constituent mineral material. Very short segments of daily training, such as a brief period of working trot or gallop over a hard surface, will rapidly load and impact the appendicular skeleton and provide enough stimulus to elicit bone response.

In the book *Equine Locomotion*, authors (Back and Clayton, 2000) point out that “as the level and duration of training increases, respective gains in performance are reduced.” There is a fine line between achieving functional adaptations and incurring pathologic problems. While mechanical stimulation through cyclic loading of the long bones might generate increased bone mass, this stimulus can have a deleterious effect on short bones, such as those in the carpus (knee). The internal architecture of these short bones provides maximal strength and shock absorption to protect joint cartilage using minimal bone material. However, inappropriate exercise can increase stiffness of subchondral bone, which lessens its shock absorption capability. Ultimately, damage to joint cartilage could lead to development of degenerative joint disease (DJD).

Problems arise in cases where exercise demand exceeds structural adaptation, leading to pathologic changes such as microfractures, bone inflammation (bucked shins), growth plate abnormalities, or joint injury, to name a few. Fatigue damage to bone, such as microfractures that occur from cumulative loading, is repaired by the remodeling process. Bone-eating cells (osteoclasts) that “excavate” an area of the defect initiate the repair, weakening the bone as bone cells are resorbed. If limb loading continues before bone production cells (osteoblasts) lay down new matrix, a stress fracture can occur.

To an extent menopausal women can modulate hormone-influenced osteopenia (low bone mineral density) and osteoporosis (reduction of bone mass) through regular exercise that mechanically loads the skeleton. Veterinarians use this same principle to rehabilitate a horse that has been on an extended rest period due to an injury that precludes functional loading with regular exercise.

Current research is under way examining the use of biochemical markers of bone formation as a noninvasive method of monitoring training effects on bone. Fluctuations in biomarkers represent subtle changes in remodeling parameters. A series of monthly blood tests measures degradation and synthesis products involved in tissue remodeling—remodeling that occurs both during training of the young athlete and as a result of injury. The objective is to differentiate biomarkers representing normal bone remodeling from those related to injury or disease. Suspicious biomarker levels suggest that more diagnostic tools, like a bone scan or MRI, might be helpful for diagnosis.

**Take-Home Message**

The equine skeletal system is a diverse and organized system of various tissue types, capable of remodeling and adapting in response to exercise and training. As you would with all organ systems, plan and provide appropriate nutritional support and conditioning for proper development and continued maintenance of health, and watch closely for abnormalities that could indicate a problem.