On Bucked Shins

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The purpose of these studies was to better understand the condition of bucked shins so that a treatment or preventive strategy could be developed/implemented to improve the health and safety of horses and decrease the costs of this condition to the racing industry. Author's address: University of Pennsylvania School of Veterinary Medicine, New Bolton Center, Kennett Square, PA 19348.

1. Bucked Shins—The Syndrome

Bucked shins seem to be associated with young horses exercised at speed. The condition may be seen in Thoroughbred, Standardbred, and Quarter Horses. It has been reported that 70% of young Thoroughbred racehorses in training develop a repetitive loading injury in their third metacarpal bone (MCIII) known as bucked shins.[1] This is a fatigue injury of bone and usually occurs in 2-yr-old horses during the first 6 mo of their training and may be seen bilaterally. If the condition does occur bilaterally, the left limb is usually involved before the right. This has been associated with horses that train and race in a counterclockwise direction and are on the left lead of their gait when in the turns. Clinically, the condition is diagnosed by physical examination using palpation of MCIII to reveal heat, pain, and tenderness, with or without swelling over the dorsal or dorsomedial surface of the MCIII. The affected animals tend to be short strided, uncomfortable at exercise, or lame. Radiographic diagnosis may be delayed from the clinical onset of signs but is evidenced by periosteal new bone formation over the dorsal or dorsomedial aspect of this bone.[1] The proliferation of periosteal new bone may be extreme in some cases (Fig. 1). Nuclear scintigraphy is a sensitive diagnostic tool but is rarely indicated with a positive history and physical examination. With few exceptions, once the condition occurs and resolves, the animals do not experience this problem again. This "vaccination" or adaptation phenomenon may be used in distinguishing animals at risk from those that are not. This observation has been used in the past to intentionally buck horses' shins to get past the problem. One downside to this method, besides the lost training and racing days, is the risk that the horse will go on to develop a stress or saucer fracture of MCIII that could lead to a catastrophic fracture (Fig. 2). Horses that buck their shins and stop training may re-buck when reintroduced to training. It is interesting to note that the condition of bucked shins occurs in young horses entering training. These animals are typically 2-yr-old horses and are equivalent to human adolescents. They are still growing and will have open physes. If by chance older adult horses are entered into training as 3- or 4-yr-olds, they may develop bucked shins as well. Racehorses that have trained and raced successfully in Europe may develop bucked shins when they race in North America on dirt tracks. It is interesting to note that horses are running on harder surfaces in North

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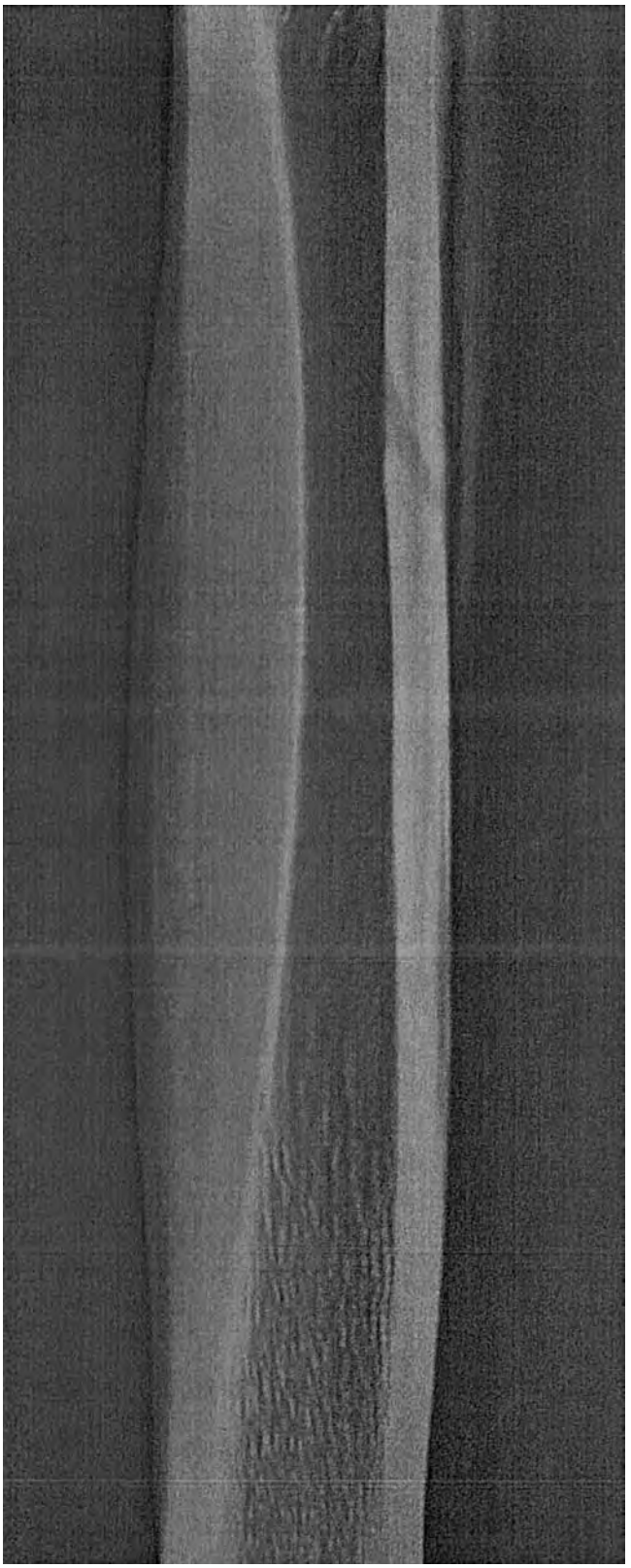


Fig. 1. This radiograph shows a clinical example of bucked shins. The periosteal reaction can be seen as an elevation of the dorsal cortical surface with the original cortex still defined.

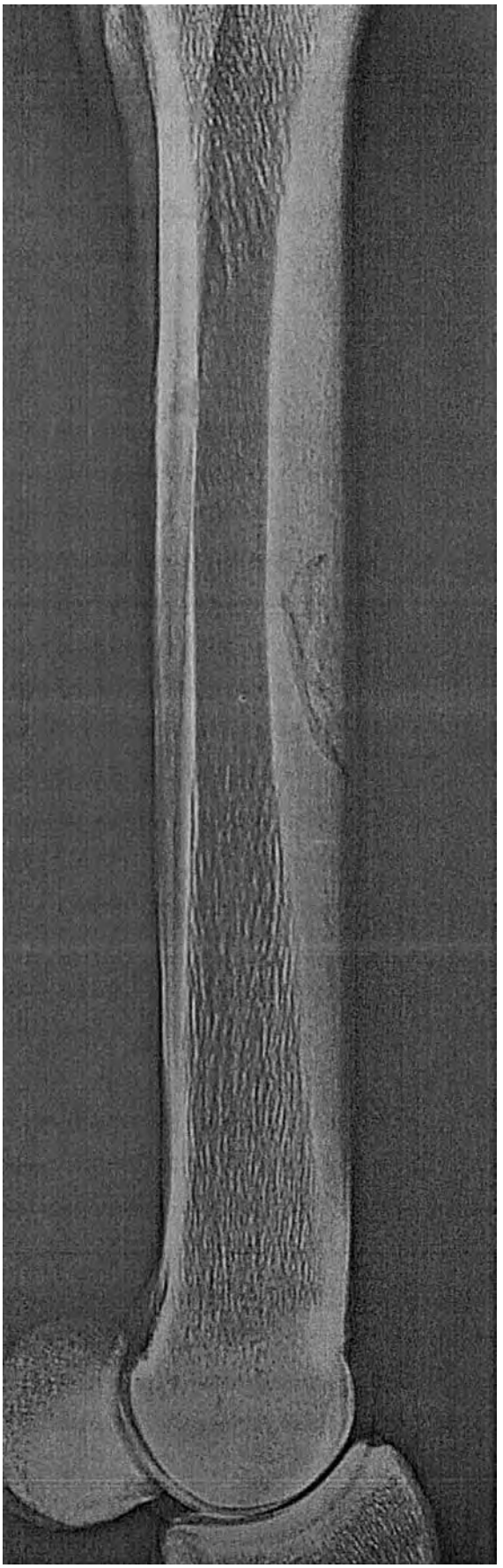
America than on the turf courses in Europe. Some animals (~12%) that buck their shins will develop a radiographically visible stress fracture on the dorsolateral surface of the MCIII up to 1 yr after the original bucked shin injury (Fig. 2).[2] Clinically, this injury is usually seen first as a fracture line with periosteal callus formation and cortical remodeling during the healing phase. However, cortical remodeling can precede the occult fracture line seen radiographically. Horses that develop stress fractures will have a previous history of bucked shins, usually with significant evidence of periosteal new bone formation at that time. Catastrophic complete midshaft fractures of MCIII can occur when these horses are exercised at speed or raced. The term dorsal metacarpal disease has been used to describe all the manifestations of these conditions.

It is interesting to note that bucked shins, while occurring predominately in Thoroughbred racehorses, is uncommon in Standardbred racehorses. These two breeds train and race at different speeds and in different gaits; the Thoroughbred training in several different gaits and racing in a galloping or running gait, whereas the Standardbred trains and races at a trot or pace, changing only the speed of travel. The Thoroughbred goes faster in its racing gait (64 km/h) than the Standardbred (48 km/h). These observations of the naturally occurring injury to bone suggest that understanding the details of mechanical loading may play an important role in determining how and why bone fatigue occurs in vivo.

2. Classical Etiology/Pathogenesis

Bucked shins as a disease/syndrome of Thoroughbred racehorses has been described for generations. The classical description of its etiology and pathogenesis was associated with other fractures of bone. Basically, the story told of subperiosteal hemorrhage and micro-fractures on the surface of MCIII as a result of high-speed exercise, concussion, and external trauma. The healing pattern was presumed to be that of secondary fracture healing with callus formation. Little observation or experimentation was performed to study the etiology or pathogenesis. Empirical judgments of pathogenesis were advanced without challenge, and classical treatments involving pin firing of MCIII and rest were used without proof of efficacy. Micro-fractures had not been demonstrated histologically. When examining this classical description, there seemed to be no relationship between the described pathogenesis and treatment. By contrast, secondary fracture healing with callus formation occurs when there is motion associated with the ends of the fracture fragments. Primary healing or direct bone remodeling, without callus formation, would be expected in this instance, because the bone was intact.

To study bucked shins, an hypothesis of a repetitive motion injury associated with high-strain cyclic fatigue was developed.



3. Experimental Studies to Determine Etiology–Pathogenesis–Pathomechanics

To elucidate a more realistic hypothesis for the etiology and pathomechanics/pathogenesis of bucked shins, a number of in vitro and in vivo experimental studies were performed to understand how bucked shins in Thoroughbred racehorses relate to fatigue failure of bone. Because Thoroughbred racehorses have a much higher incidence of bucked shins compared with Standardbred racehorses, a starting point was to compare the in vitro fatigue properties of Thoroughbred MCIII to those of the Standardbred.

To accomplish this study, 26 dumbbell-shaped specimens machined from MCIIIs of five adult Thoroughbreds and 25 specimens machined from MCIIIs of five adult Standardbred horses were tested in fully reversed cyclic bending experiments, using a constant strain rotating cantilever model that measured load decrement.[3] All specimens were tested fresh, and tests were completed within 3 h after the death of the horse. All tests were performed at 40 Hz, after a pilot study examining the differences among 10, 20, and 40 Hz showed no significant differences in fresh bone. Testing of all specimens continued until the specimen broke or had a 30% loss of stiffness. Three different offsets (deformations) were used to establish nominal strains of 7500, 6000, or 4500 micro-strain in the specimens.

Data were analyzed using a power regression model for each horse and for each breed. Statistical differences were not found among the curves for individual horses of the same breed or for the curves between the two breeds. Pooling the data resulted in a data set that described the in vitro fatigue characteristics of cortical MCIIIs from Thoroughbreds and Standardbreds, greater than 4 yr of age, subjected to fully reversed cyclic loading (Fig. 3).

In vitro fatigue studies of equine MCIII have been the subject of several papers in the literature. Gibson et al[4] described the fatigue behavior of equine MCIII using rectangular beams of cortical bone (10 × 4 × 100 mm), tested monotonically and in fatigue in four-point bending. Deformations reaching 10,000 micro-strain (1% strain) were used for the fatigue tests. The beams were machined from the four quadrants of the bone in the mid-diaphysis. The animals were between 2 and 5 yr old, and two were in race training and the other four had raced numerous times. The authors reported a loss of modulus in the medial and lateral regions but not in the dorsal regions as the fatigue tests approached failure. The SDs of the fatigue data were consistently higher than the means in the dorsal

Fig. 2. A stress fracture (saucer fracture) is present in the distal third of the dorsolateral cortex of MCIII in this radiograph. These fractures seem to only occur in horses that have previously bucked their shins.

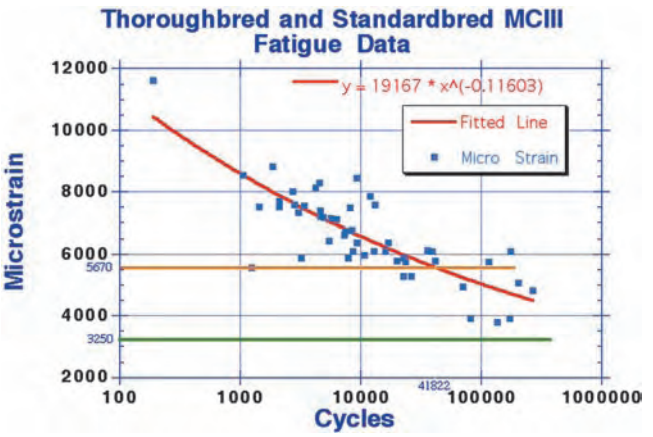


Fig. 3. Fatigue data is presented using strain versus number of cycles. The Thoroughbred and Standardbred data are combined because they were not different. Superimposed on this data set is the average in vivo strain recorded for the young Thoroughbred horses in training (5670 $\mu\Sigma$) and the older horse (3250 $\mu\Sigma$). It can be noted that the young horses strain levels intersect the in vitro fatigue regression line at 41,822 cycles, whereas the older horse does not intersect this data set until somewhere after 1,000,000 cycles.

region, which may have been related to the histories of the specimens that included two horses in race training and the remaining horses having raced. The authors provided a good discussion of problems associated with in vitro fatigue testing, i.e., temperature variation, cyclic rate, and loading methodologies. They concluded "in vitro laboratory fatigue testing does not account for the in vivo biological responses to fatigue damage."

The same group evaluated the residual strength of equine MCIII following in vitro fatigue loading.[5] Using the same methodology, they cycled bone specimens from 0 to 5000 micro-strain for 100,000 cycles and then compared their strength in bending with matched monotonically loaded specimens from the same individual. They showed that the modulus of the specimens did not degrade over the 100,000 cycles tested. The residual strength was only 3% lower in the cycled specimens versus the monotonically loaded ones. Using this same model once more, this group looked at remodeling and microcrack damage created in vitro in monotonically loaded specimens and those subjected to cyclic fatigue at 10,000 micro-strain.[6] After failure, they bulk-stained the specimens in basic fuchsin, and 100-μm cross-sections were cut and examined microscopically. Two types of cracks were seen. The unstained cracks seen in woven bone were thought to be damaged Sharpey's fibers; their length increased after failure. The stained cracks were larger and were seen near the fracture surface and on compressed surfaces. They were more numerous in specimens with a higher modulus and a shorter fatigue life. Prior remodeling of bone did not seem to influence the presence of microdamage in these studies.

Because in vitro fatigue data of equine MCIIIs showed no difference by breed, it seemed that other factors were important in the etiopathogenesis of fatigue failure of bone in the Thoroughbred racehorse. Because fatigue of structures can be associated with material properties or section properties, the sectional properties of the two breeds' MCIIIs were examined. To see if the bone's inertial properties were different, 30 pairs of second, third, and fourth metacarpal bones of the racehorses were examined (10 Standardbred and 20 Thoroughbred horses).[7] Data on the Thoroughbred bones were grouped by age into five groups: yearlings, 2-yr-olds, 3-yr-olds, 4-yr-olds, and "aged" horses (older than 5 yr of age). The Standardbred data was divided into two groups with five pairs in each group: yearlings and "aged" horses. Comparisons were made between breeds of a particular age group and between the age groups of a particular breed. Mean section properties were plotted against percentage of bone length to observe patterns proximal-to-distal for each property measured.

Results of this study showed that age and breed were the only factors affecting section properties, i.e., there were no left/right differences. In the Thoroughbred group, all section properties were much lower for yearlings than for any other age group. Little differences were seen in cross-sectional areas in horses over 2 yr of age. Changes in the second moments of area that relate to bending stiffness in a particular direction did show significant differences. These second moments related to bending in the dorsopalmar direction and the medial-to-lateral direction were used to determine the principal moments I_{min} and I_{max}. The most significant changes in the bone occurred at the midsections between the ages of 1 and 2 yr, but continued change occurred until age 4 yr. No observable changes took place after 4 yr of age. I_{min} was smaller in the yearling Thoroughbred than the yearling Standardbred but was larger in the adult Thoroughbred than in the adult Standardbred (Fig. 4); therefore, the Thoroughbred changed this property to a greater extent than did the Standardbred during the first 2–4 yr of life, just when the animal is at risk for bucked shins. One could hypothesize that the inertial property I_{min} changed as the horse underwent training and enlarged to reduce strain or deformation in the bone in dorsopalmar bending. As the animal got older and increased its inertial properties because of the training, the deformation of the bone (strain) would decrease with the same applied loads.

In a classic publication in 1979 Goodship et al[8] showed that the strains (deformation) in the forelimb of the pig would double by resecting the ulna and allowing the pig to run around on its radius. By the time the radius and healing ulna regenerated the same area of bone in the cross-section, the

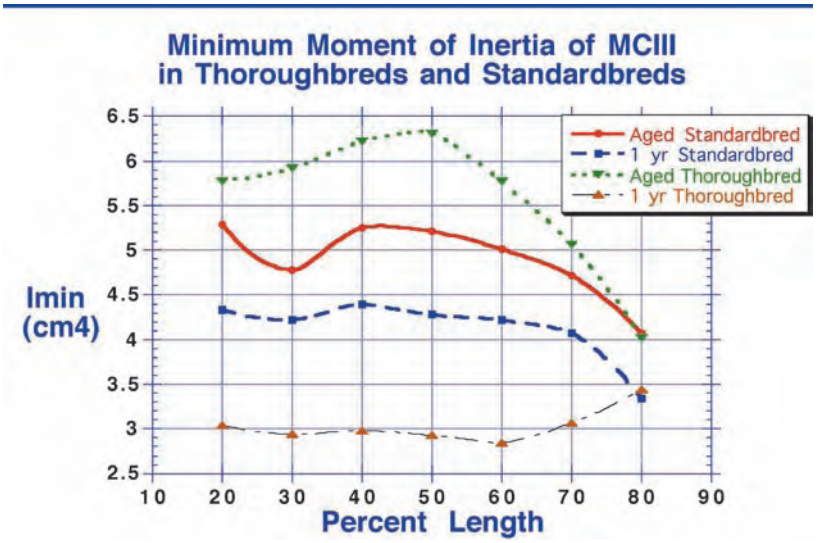


Fig. 4. Graphical representation of MCIII inertial properties relating to dorsopalmar bending of the MCIII is demonstrated by breed and age. It can be noted that the Thoroughbred racehorse increases its I_{min} to a much greater extent than does the Standardbred racehorse. Horses would seem to be at risk for bucked shins during this change in shape.

strains would return to normal. The authors did not look at inertial properties but used cross-sectional area. This same phenomenon may be at work in the horse with young small-diameter bones that is being asked to carry a larger load during high-speed training. The inertial properties must increase to handle that load.

The next step was to record in vivo bone strain on the MCIIIs of horses training for racing at or near racing speed. To determine whether differences existed between young (2-yr-olds) and older established racehorses, four 2-yr-old horses were purchased and trained for approximately 6 mo.[9] A veteran 12-yr-old Thoroughbred racehorse that had raced more than 40 times was also used in this study. All the horses were trained by professional trainers and were exercised with rosette strain gauges mounted on their MCIIIs on a dirt racetrack, and all but one horse raced before strain gauge measurement. All animals were exercised at work (breeze), i.e., their racing gait at racing speed. Speed was monitored with a stopwatch using furlong poles as distance markers. An onboard tape recorder captured the data, and strain measurements were made continuously throughout the workout. One channel of the tape was used to record a voice overlay of the jockey during the workout to fit the gait patterns to the bone strain. All horses were urged to their maximum effort for one quarter of a mile, which included the stretch run.

In vivo strains measured during these experiments using the rosette gauges were resolved using a graphical engineering solution known as Mohr circle analysis and reported as principal strains and directions when possible. The four 2-yr-olds recorded peak compressive strains: 1) −4761 micro-strain, 2) −4533 micro-strain, 3) −5670 micro-strain, and 4) −4400 micro-strain (−4841 ± 572 micro-strain). The 12-yr-old racehorse recorded strains of −3317 micro-strain. Horse number 3 developed clinical signs compatible with the diagnosis of bucked shins. His strain gauge measurements were approximately 6 SDs above the average maximal strains of horses 1, 2, and 4 (−4565 ± 182.6 micro-strain).

Changes in speed from a trot to the racing gallop changed the principal strain direction by more than 40 degrees on the dorsolateral surface of MCIII at the site of strain gauge placement in all horses studied. Animals that were trotting showed tensile strains in the long axis of the bone on the dorsal/dorsolateral surface of MCIII. At racing speeds, this same surface of the bone showed compressive strains.

Following the acquisition of in vivo strain data from these young horses, an attempt was made to correlate this data with the in vitro fatigue data previously generated by determining the average number of cycles that a young Thoroughbred racehorse would gallop in training before the onset of bucked shins. To accomplish this, the records of six 2-yr-old Thoroughbred racehorses in training that had developed bucked shins were examined to establish the distances the animals trained before the onset of fatigue failure of bone.[3] To determine the number of cycles galloped over these distances, six 3-yr-old racehorses were exercised at a canter, gallop, and at work (racing speed) to evaluate the length of stride in each of these gaits. The stride length was then divided into a mile to determine the number of strides (cycles) per mile. The total number of gait cycles of the six animals under study was

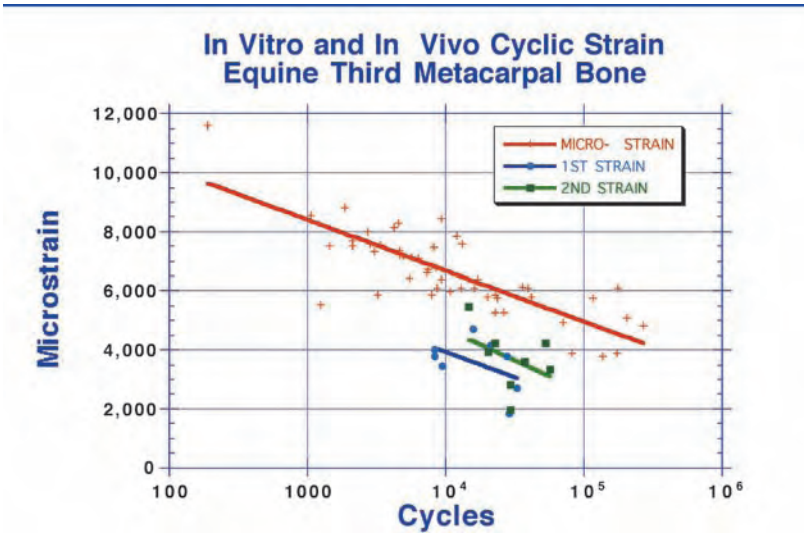


Fig. 5. Superimposition of the in vivo strain measurements of horses at two different times in training over the previous in vitro fatigue data shows that the bones (regression lines) of the horses in training are moving toward the in vitro fatigue curve. This increased risk event occurs when the horses should be changing their inertial properties to "make them safe."

estimated based on the distances covered in a canter, gallop, and at work. These horses were trained in these gaits between 10,000 and 12,000 cycles/mo. The six horses were diagnosed with bucked shins between 35,284 and 53,299 training cycles.

Superimposition of the in vivo strain gauge data from the four young Thoroughbred horses and the older Thoroughbred racehorse on the in vitro fatigue data previously compiled, along with the in vivo number of cycle data for the incidence of bucked shins in the six Thoroughbred racehorses, showed an almost exact correlation (Fig. 3). Furthermore the superimposition of the 12-yr-old horse on this data showed that older horses would not reach the critical strain-number of cycles line of the fatigue curve for more than 1,000,000 cycles. These 80-plus months should give the bone adequate time to remodel without being at risk. Older horses do not train as much as younger horses as they continue to race. The very high strains shown in the instrumented horse that bucked its shins compared with the age-matched horses that did not buck demonstrated a change in either material or section properties that allowed these larger deformations.

To evaluate changes in whole-bone stiffness and changes in local surface strains over time with superimposed training, 12 yearling Thoroughbred racehorses were purchased at auction and divided into four groups.[10] Three horses were kept as controls and allowed free exercise at pasture. The other nine horses were trained in groups of three per year by a professional trainer using a classical training regimen. Complete training records were kept, and the strain on each horse's MCIII was measured using rosette strain gauges at two different times. The right MCIII was strain gauged after each horse was broken to saddle and able to gallop a mile (1.5 km) at 13.4 m/s. The left MCIII was strain gauged several months after the right side, well into the training process. Use of a radar gun and radar detector marking the tape recordings allowed comparisons of the two different strain-gauge sessions at the same speed. Maximum principal strains were compared between the first and second strain-gauge sessions, where possible, to determine the effect of training on measured bone strain. One horse developed bucked shins after the first strain gauge session and was too lame to be instrumented a second time. Another horse had equipment problems and was only measured once. Seven horses had both sessions recorded, and four of these horses increased their bone strains from the first to the second session (mean increase 1384 $\pm$ 819 microstrain; Fig. 5). Two horses showed a decrease in bone strain from the first session to the second, and two horses were essentially unchanged. It is interesting to note increased strains in some individuals when one would expect decreasing strains based on increasing inertial properties (section properties) of the bone. If the change in inertial properties did not occur then strains would be expected to remain the same; conversely, if material properties degrade (modulus) then the strains would be expected to rise. Measurements of whole bone stiffness and/or bone material properties from animals in the normal population and in animals trained for racing might delineate the changes associated with increasing strain measurements in the face of continued training. Whole bone stiffness was measured in 40 pairs of MCIIIs from animals 2 mo to 28 yr of age, using non-destructive three-point dorsopalmar bending tests in vitro.[10] Bone stiffness measurements were calculated from load displacement values obtained using a jig and clip gauge assembly mounted on each

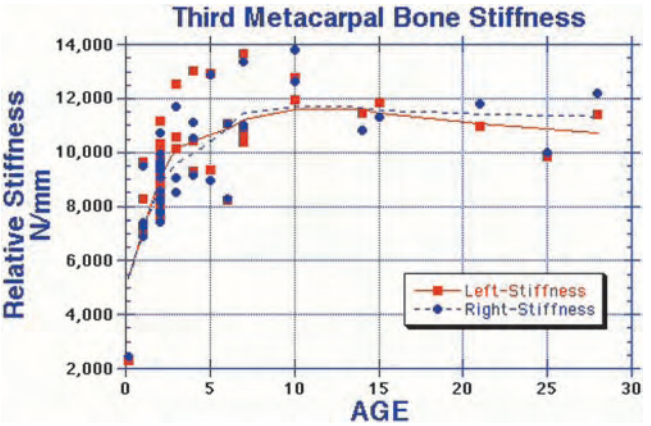


Fig. 6. Paired whole bone in vitro stiffness measurements show increasing bone stiffness for the first 5–6 yr. Animals in different stages of training make up the group between 2 and 5 yr where large differences are seen. The 3-yr-old group shows differences within pairs that can be seen graphically.

bone coupled to the load cell of an Instron 1331 testing machine. The bones showed general increases in stiffness until they reached a plateau at about 6 yr of age (Fig. 6).

The intact MCIIIs of 12 experimental 2-yr-old Thoroughbred racehorses were included in these tests using non-destructive three-point bending in vitro, after the in vivo portion of another experiment. Paired bone stiffness measurements showed dramatic changes in three of the trained horses, with differences between right and left MCIIIs of 16, 27, and 23%. One of these horses bucked its shin between strain gauge sessions and was not strain gauged the second time (see above). The other two horses had clinical evidence of bucked shins. Three other trained horses showed differences in bone stiffness in the 6–8% range, and three trained and three control horses showed no difference between their right and left limbs.[10] These studies showed that paired limbs from young Thoroughbred racehorses in training might not have the same mechanical properties. The dramatic change in stiffness noted in three of the horses versus no change in the controls was presumed to be related to the training on an oval track. The animals train in a counterclockwise direction, making the animal use his left leg as a lead limb in the turns. Thus, the changes in the bone are graded so that changes occur first in the left forelimb and then the right. The natural history of bucked shins shows that the left is usually involved and it precedes the right when both are affected. The enigma presented by the data was that the left leg in two of the three horses was stiffer than the right, whereas the third horse had the right leg stiffer than the left.

Besides being a model for fatigue failure in bone, bucked shins in the Thoroughbred racehorse represents a significant clinical problem for the horse racing industry. Therefore, understanding the etiology and pathogenesis of this condition should help point to a method of treatment or prevention. Experimental studies showed that the inertial properties of Thoroughbred racehorses increase dramatically at the same time when growth and superimposed training leads to bucked shins. This would seem reasonable because horses that do not train for racing do not develop these same inertial properties. To prevent bucked shins, it would seem necessary to change the inertial properties early in the training period. In addition it was shown that the dorsal surface of MCIII is under tension when the animal is training in a slow gait and changes to compression when the animal races. Classical training methods for Thoroughbred racehorses consist of long slow gallops with the racing speed and gait used sparingly. Therefore, horses that train in a classical manner with tensile forces on the dorsal surface of the MCIII would be well suited for training but may not be so for racing. Horses that mimic racing in their training may be more suited for racing since the dorsal surface of the MCIII will be loaded in compression. It seems obvious that bone that models and remodels in response to tensile forces on the dorsal aspect of MCIII will be poorly adapted for large compressive forces that are seen during racing. High-strain cyclic fatigue (bucked shins) will and does occur quickly in the young training Thoroughbred racehorse usually about the time of the first start when the animal is running in its racing gait.

The classical hypothesis of subperiosteal hemorrhage and micro-fractures seems to be the wrong interpretation of the bone's morphology. The high-strain events of bending of the MCIII induce bone formation on the dorsal surface of MCIII. Classical training will induce this bone on the dorsomedial surface. Lamellar bone formation can accrue on surfaces up to 1–2 microns/day. Faster bone formation may occur using fiber bone that forms the periosteal new bone that is vascular and porous. This normal periosteal bone formation is interpreted by the clinician as bucked shin, and therefore, disease. The new bone formation is not disease per se but an appropriate response of bone to high-strain repetitive motion injury of MCIII. Therefore, it is this injury that needs to be addressed.

This brings us to methods of prevention. Because exercise is the problem, a change in the pattern of exercise may also be the solution. Our hypothesis that training that mimics racing should produce a bone structure that may be helpful in decreasing the incidence of bucked shins by changing the inertial properties was tested. With the understanding that slow speed gaits produce tensile strains on the dorsal surface of MCIII, whereas high-speed exercise induces compressive strains in this same region, a study was undertaken to determine the effects of different training regimens and track surfaces on the modeling and remodeling of MCIII in the Thoroughbred racehorse.[11]

Eight 2-yr-old Thoroughbred horses were purchased at auction before any training and were divided into four groups of two horses each. Classical training methods were used for the horses in groups I and II. Group I horses trained on a dirt track only, whereas group II horses trained on a wood-chip track only. Group III horses (control group) were not trained but were allowed free exercise in a 5-acre pasture. Group IV horses were trained using a modified classical training program on a dirt track only.

The classical training program was comprised of daily gallops (~18-s furlongs, ~11.2 m/s) of 1 to 2 miles/day (1.6–3.2 km) followed by shorter "works" or "breezes" at racing speed (~14-s furlongs, ~14.4 m/s) once every 7–10 days and increasing in distance from two to six furlongs (0.4–1.2 km) progressively over the course of the study. The modified classical training method used similar daily gallops, but the frequency of the high-speed "works" increased to three times a week, whereas distances increased progressively from one to four furlongs (0.2–0.8 km). The study was completed over a 5-mo period, with groups I and IV being trained on a dirt track and group II horses trained on a wood-chip track.

Following 5 mo of training or pasture turnout, the animals were euthanized, the MCIIIs of all horses collected, and cross-sectional inertial property measurements made using methods described previously.

Examination of the inertial properties of the 50% sections of MCIIIs from the different groups showed that the minimum principal moments of inertia (I_{min}) of groups I and II were similar to group III horses that did not train at all. The principal moment, I_{min}, of group IV horses was greater than the other groups and was similar to the I_{min} moments previously reported for mature racehorses (Fig. 7E).

Gross evaluation of the microradiographic cross-sections from the mid-diaphysis of MCIIIs from these horses showed that bone remodeling occurred only medially and laterally in groups I and II. The filling of secondary Haversian systems with new bone was more complete in group I specimens, indicating that the remodeling process was further advanced in the group that was exercised on the harder dirt track surface. There seemed to be a distinct lack of remodeling activity in the dorsal and dorsolateral regions of these sections in groups I and II. Group III and IV horses showed extensive remodeling throughout the cortex. Groups III and IV specimens revealed remodeling in the dorsal and dorsolateral aspects of the bone that was not seen in groups I and II.

Interpretation of the data from this experiment suggested that horses that train on a harder track surface seemed to remodel their bone at a faster rate than horses that exercised on a more compliant surface. Previous studies have shown that classically trained horses that exercise on a hard surface seem to have a higher incidence of bucked shins than horses training or racing on a more compliant surface.[12] One horse in group I bucked its shins during the training period. Classical training methods applied to horses training on a hard or soft track did not effectively change the inertial properties (I_{min}) that influence bending of MCIII in a dorsopalmar direction. In contrast, exercise regimens (group IV) that stressed MCIII in compression on its dorsal surface did change I_{min} in a significant manner, consistent with adult racehorses evaluated previously that were no longer at risk for bucked shins.

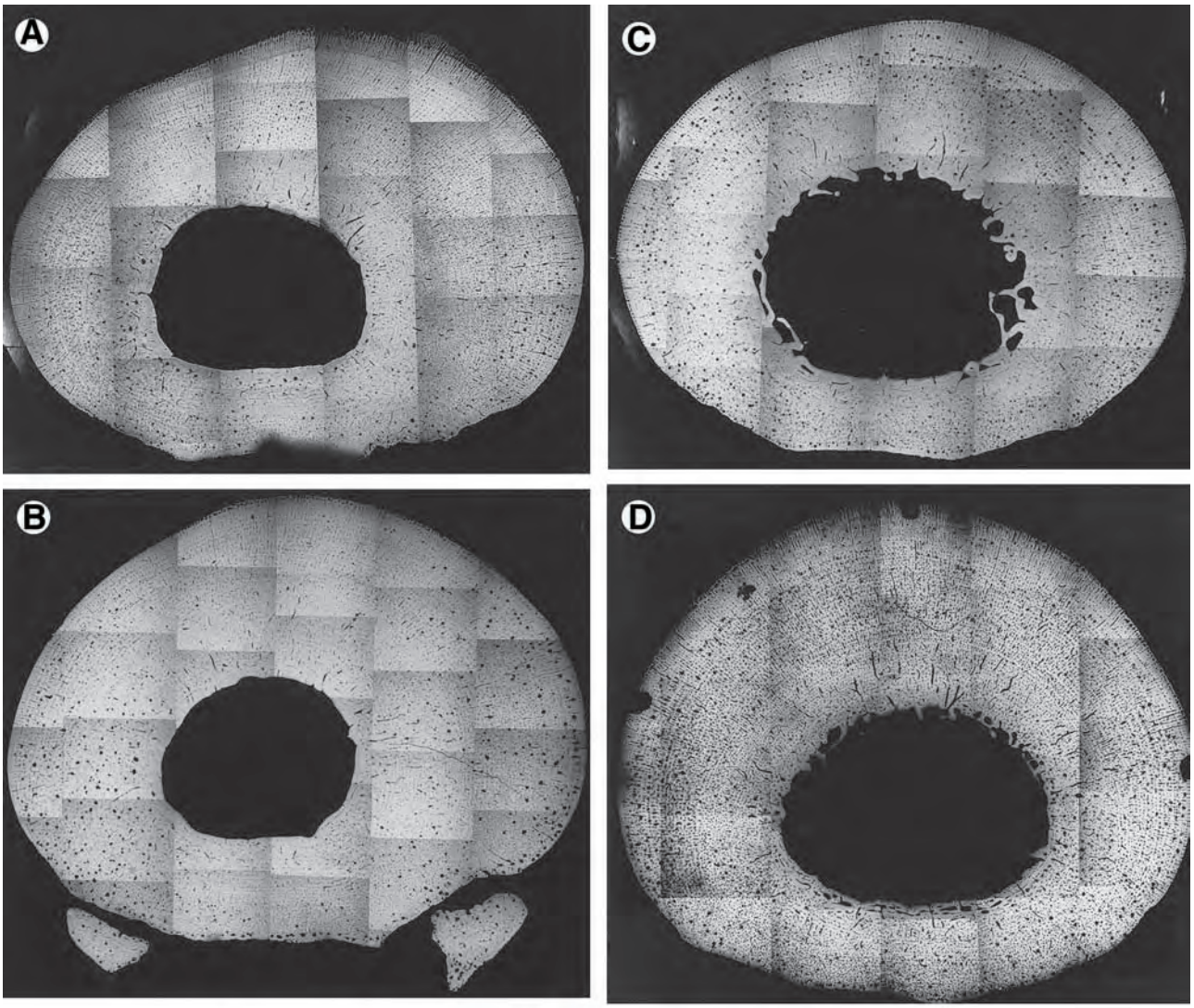
The results of this study supported the concept that exercise could be designed to optimize the shape of MCIII. This, in turn, should influence (decrease) the incidence of bucked shins in this Thoroughbred racehorse model, and therefore, the problem within the industry.

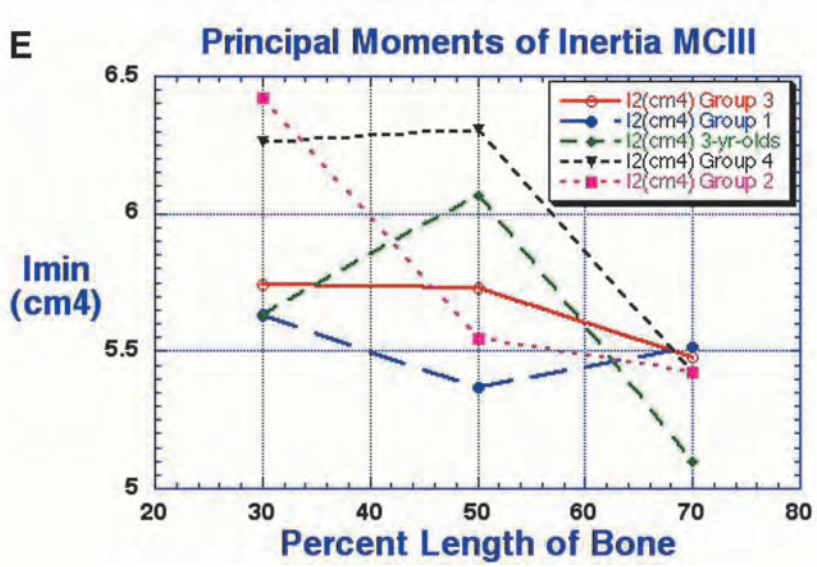
To prove the efficacy of such an hypothesis it was necessary to design and carry out a large study of young Thoroughbred racehorses in training using different training modalities.

Based on these previous experiments, a training program was designed around the modified classical regimen.[13] To determine the efficacy of this adaptive training program to decrease the incidence of bucked shins, a prospective and retrospective study was started using five commercial training stables. Two of the stables (2 and 5) were aware of our modified classical training program and were using it as a basis for their training regimen. The other stables (1, 3, and 4) were thought to be training in the classical manner. Stables 1 and 2 used the same racetrack for training. Stable 4 trained on a racetrack as well, whereas stables 3 and 5 trained on their own farm training tracks.

To be a part of this study, trainers needed to keep complete daily records of each horse's training with accurate accounts of distances the animals jogged, galloped, and breezed. These distances were collected and used in the data analysis as rates (miles per week). In addition, physical exams and indications of events not related to bucked shins were recorded. Horses entered into the study had to be 2-yr-old Thoroughbreds being entered into race training for the first time. The animals were followed for 365 days after training began. Data collection stopped when the horses bucked their shins, were sold, or stopped training because of another event not related to bucked shins. The study included 11 yr of training data from 226 2-yr-old Thoroughbreds, but all years were not represented by all stables. Of the 226 horses 56 bucked their shins, and the other 170 either completed the 365-day observation period or were sold while in training.

Using STATA statistical software,[14] regression analysis and survival analysis techniques were used to explore the data. Using the log rank test, we found a significant difference ($p < 0.05$) in survivability of the 2-yr-olds at the five stables. Because stable 2 had the best survival and stables 1 and 4 had the worst, evaluation of the relationships between jogging, galloping, and breezing between





these stables was carried out. Stable 2 with the highest breezing rate had the lowest incidence of bucked shins, whereas stables 1 and 4 had the highest galloping rates and the highest incidence of bucked shins.

The next step was to explore the significance of dependence of survivability on each of the three training activities. This was accomplished using a Cox regression stratifying on stable. Because jog rate was not statistically significant ($p = 0.113$), the Cox regression was repeated without the jog rate data and showed that gallop rate ($p = 0.005$) and breeze rate ($p = 0.007$) were significant. The hazard ratio for this data set showed that galloping (1.364625) increased the likelihood of bucked shins by 36.4%, whereas breezing (0.0140464) was protective, reducing the likelihood of bucked shins by 98.6%.

4. Discussion

Twenty years of evolutionary experiments, based on an initial observation of a marked difference in the incidence of fatigue fractures between two different breeds of racehorses, has led to a more complete understanding of a natural model for fatigue failure of bone. We now can compare in vitro and in vivo fatigue behavior and observe bone adaptation to different exercise regimens. Adaptive exercise has been shown to change the geometric properties of MCIII, to influence bone modeling and remodeling, and to reduce the incidence of fatigue failure of MCIII.

Comparisons of the Thoroughbred with the Standardbred racehorse show major changes in inertial properties of MCIII as a result of growth and superimposed training. Comparisons of young Thoroughbreds that are susceptible to fatigue failure (bucked shins) with older resistant animals suggest that changes in bone inertial properties are an important factor affecting the incidence of this injury. Large MCIII I_{min} values reflect probable increases in MCIII stiffness in the dorsopalmar direction, and thus, reduced peak strain during high-speed exercise. The inertial properties of the proximal tibia have been shown to be predictive for the development of fatigue fractures in military recruits,[15,16] just as the inertial property measurement of the 5-yr-old Thoroughbred MCIII shows that the animal is no longer at risk for bucked shins.

In vivo strain measurements of Thoroughbred MCIIIs demonstrated higher peak strains under physiological loading than previously reported in any animal species. Although all in vitro test conditions differ from the in vivo loading, most involve significant bending components that can be expected to produce accumulated fatigue failure in composites such as bone. Therefore, whereas the in vitro data may not produce the in vivo intrinsic fatigue mechanism, the superimposition of the in vivo strains reported for the young and older horses at racing speed produce a striking predictive relationship for risk of developing this fatigue injury (Fig. 2).

Large surface strains, measured in vivo at high speeds on the dorsal aspect of MCIIIs between the extensor tendons in young 2-yr-old Thoroughbred racehorses in training, contrast dramatically with the smaller strains measured in adult racehorses that have raced successfully. Strains, under a given load regimen, measured on the surface of bone, relate both to the bone's modulus and to its inertial properties. Because inertial properties have been shown to increase with age, and bone strains during high-speed exercise have been shown to decrease in older horses, it was hypothesized that changes in bone inertial properties and/or modulus serve to lower the peak bone strains as the young racehorse matures. However, it was seen that classical training techniques did not change the inertial properties, therefore leaving the animals at risk for bucked shins. We observed that a certain percentage of young animals actually increased MCIII surface strains after several months of training. This may have been related to increasing speed during training or decreasing material properties of the bone. Whole bone stiffness measurements showed right-to-left differences of up to 27% in horses in training, whereas there was never a right-to-left difference in the non-trained control animals. Because bucked shin occurs bilaterally but sequentially in young Thoroughbreds, usually on the left side before the right, it is possible that the developmental stiffness changes in limbs are not synchronized but may respond to the predominance of the left lead used by the horse in its racing gait, in the turns as the horse travels in a counterclockwise direction around the racetrack. Maximal strains at exercise and bone stiffness parameters probably both change with time and may be declining on one

Fig. 7. A montage of microradiographs from the mid-diaphysis of MCIII is shown for four different horses subjected to four different exercise programs. A, group 1 horses trained in a classical manner on a dirt track. This horse bucked its shins, which can be seen as periosteal new bone formation on the dorsomedial cortex. B, group 2 horses trained in a classical manner on a wood-chip track. C, group 3 horses were control horses and were just turned out to pasture. D, group 4 horses trained in a modified program that included breezing three times/week. E, inertial properties of horses trained using these different regimens show that classical training (bottom two lines, groups 1 and 2) has no effect when compared with horses that do not train (middle line, group 3). Modified training that includes short distance fast exercise (high strain cycles) provides changes (top line, group 4) that equal or surpasses adult trained horses (second line) that have been previously reported.[7]

side while increasing on the other. Increasing bone strain measured at high speed during training, as seen in four of the seven Thoroughbreds reported, is suggestive of rapid bone stiffness change in vivo because of exercise.[10] Several possible related explanations exist for this observation: 1) bone stiffness decreases in vivo, much as it does in vitro, when the bone undergoes cyclic fatigue, and 2) bone stiffness increases because of inertial property changes in MCIII but may be overwhelmed by decreasing intrinsic material stiffness.

If Wolff's law is strictly applied, it follows that a bone that adapts to a particular peak tensile strain may not be adequately prepared to resist far larger peak compressive strains in the same location.

A recent in vitro fatigue study of equine MCIIIs showed a difference in fatigue resistance to bending loads in different anatomical quadrants.[4] Bone that was loaded in bending around the physiologic bending axis had greater fatigue resistance than bone bent at 90 degrees to this axis.

We hypothesized that, to adequately adapt for racing, the MCIII should be exposed during training to strains of the actual magnitude and direction experienced during racing. Furthermore, the high incidence of bucked shins in Thoroughbreds suggested that loading to produce such peak strains and concomitant adaptive remodeling did not occur in a large number of Thoroughbreds in classical training programs before racing.

Previous in vivo studies, using the functionally isolated rooster ulna, have shown that low numbers of loading cycles (four per day) were adequate to maintain bone mass.[17] Thirty-six cycles were enough to stimulate bone formation in this model, but again, the loads, although still physiologic, were such that the bone was loaded in a different direction. The resulting periosteal new bone formation that was seen in this model is the same type of bone reaction observed in the Thoroughbred racehorse MCIII with a fatigue injury.

Taking these observations into account, an exercise (training) regimen was developed that modestly increased the small numbers of high load cycles using peak load magnitudes and directions that are seen during racing. Increasing the number of short distance works (breezes) from once every 7–10 days, as it occurs with classical training programs, to three times a week, produced large changes in the modeling, remodeling, and inertial property measurements of MCIII. Classical training produced little progressive change in the inertial properties of MCIII, seemingly no better than no training at all, whereas the new modified training program showed inertial property development that equaled or surpassed that observed in established older Thoroughbreds, those horses apparently no longer susceptible to bucked shins.

The idea of using exercise to produce adaptive bony remodeling is not new. Woo et al[18] showed that 12 mo of exercise in young pigs produced dramatic changes in the femur, increasing cross-sectional area by 23% and inertial properties (I_{min}) by 27% without intrinsic bone property changes. Also it has long been known that the dominant humerus of tennis players has superior inertial properties to that of the non-dominant side.[19] Both of these studies point to the benefit of adaptive exercise, but like others,[20–23] were observational and not related to efforts to produce a specific modeling/remodeling outcome. Most recently, Milgrom et al[24] has looked for exercise that can adapt bone and showed that playing basketball for 2 yr or more was protective in reducing the incidence of bone fatigue failure in military recruits.

To prove efficacy that adaptive exercise could be used to reduce the incidence of fatigue injuries of bone, an 11-yr longitudinal study of 226 commercially trained Thoroughbred racehorses in five stables was undertaken. Two of the stables (2 and 5) were aware of the modified training program and carried it out to some extent. Survival analysis of the data showed the influence of weekly jogging, galloping, and breezing rates (miles per week) on the incidence of bucked shins. The hazard ratios for this data set indicated that galloping (1.365) was a training risk for bucked shins, whereas breezing (0,014) was protective at the distances used. The winter of 1994 brought severe ice storms to the northeast. Stable 2 could not train their horses using the modified program because of the weather conditions, and they reverted to the standard classical program. This then became an unintentional cross-over design experiment, and 62% of the horses trained that year bucked their shins. Without using the 1994 year, only 9.3% of the horses trained bucked their shins in the 5-yr training period when the modified training program was implemented. Interestingly, stable 1 had 50% of the horses in training buck their shins in 1994 compared with their average not including the 1994 year of 41.3%. These two stables are easily compared with each other as they both used the same training facility and would have the least confounding variables.

When evaluating the hazard ratios it is necessary to point out that the breezing rate was much lower than the galloping rate (miles per week). These hazard ratios were based on this data set and arbitrarily increasing the breezing rate, because it is protective, would also change the hazard ratios. Certainly long distance breeze rates would be detrimental and have been described.[25]

Adaptive exercise has been shown to change the geometric parameters of a specific bone in a way that would be expected to reduce fatigue damage, whereas at the same time, significantly reduce the clinical incidence of apparent fatigue injury of this bone. This correlation, although not explicit proof of the interrelationship between the factors measured, is convincing and has already been incorporated into the racing industry on a limited scale to improve the health of this working animal.

5. Training Regimen to Prevent Bucked Shins

In 1991, Moyer and Fisher presented a paper at the AAEP annual meeting that described the incidence of bucked shin in horses training on different track surfaces and a new training regimen designed to decrease the incidence of bucked shins. This seminal paper on training efforts to reduce the incidence of bucked shins presented a rather complicated training program for starting young Thoroughbreds on the racetrack. This original training program was based on the ideas and experiments to that date that have been reviewed in this paper. Since 1991, additional experiments have been performed, and evaluation of commercial training programs has shown the benefits and drawbacks of breezing and galloping as a part of the training program. Dr. John Fisher has continued tuning his training program at the Fair Hill Training Center in Fair Hill, Maryland. Bucked shins are no longer a problem in his racing stable with horses that he started training as 2-yr-olds.

The present program assumes that the young horse is broken to ride in the fall and is able to gallop 1 mile (18–20 s furlongs) by the end of December. The training program starts in January and can be broken into three stages. The principal involved is that the horse's bones need to see the strain environment of racing as soon as possible so that bone modeling and remodeling can begin in a timely manner. The training program is 6 days a week with Sundays off. The horses walk to and from the racetrack. On the track the horse walks one-half mile and jogs one-half mile to warm up. The horse then gallops 1 mile.

Stage 1

Using the above strategy, the horse will finish the gallops two times a week with a one-eighth mile open gallop in 15 s. This speed work is done on Tuesdays and Saturdays. This speed and distance of the open gallop is repeated 10 times (5 wk).

Stage 2

After 5 wk of stage 1 gallops, the horse moves up in distance so that two times a week (Tuesday and Saturday) the horse will finish its gallop with a one-quarter mile in 30 s (15-s furlongs). This speed work is repeated 10 times, which takes up the next 5 wk. All open gallops in stage 1 and stage 2 are at the end of the gallop and are included in the 1-mile gallop.

Stage 3

After the completion of stage 2, the animal continues its training using speed work once a week (Saturday), breezing one-quarter mile in approximately 26 s (13-s furlongs). This is repeated four times (4 wk). In stage 3, the daily gallops are extended to one and one-quarter miles twice a week. After the fourth week the one-quarter mile breeze is continued with a strong "gallop out" for an additional furlong. This makes the three furlong total about 40 s. This is done for an additional 3 wk, giving stage 3 a total time of 7 wk.

After stage 3, these horses have effectively established their MCIII's shape and architecture for the longer high-speed workouts necessary for racing. They can now go on to four- to six-furlong works as needed to develop their other body systems to complete fitness for racing.

The total time of this initial training program is 119–147 days, depending on the availability of a race for the individual. This does not include any downtime for sickness or injury. Gate work is started early and often in this program. The animals are introduced to the gate in January as the program starts. All young horses are turned out in a small paddock for 1–1.5 h of exercise before daily training. This training program has shown no increase in the injury rate of young horses. An excellent by-product of this training program is the mental development of these 2-yr-olds. Because of the very relaxed atmosphere of walking to and from the racetrack, these individuals exhibit no anxiety about their work. For this training program to work the rider cannot be in a hurry to get back to the barn and on the next horse. The 2-yr-olds are not anxious about speed work because it has been in their weekly schedule since the beginning of training. All the animals walk back to the barn. Walking is a great exercise that does not seem to negatively influence bone modeling or remodeling.

Horses that develop respiratory disease during training and are off for 10 days are backed up about 10 days in their training program. Shin sore horses are treated for soreness with phenylbutazone and ice water and are walked until soreness is gone. These horses then get put in an abbreviated program where the breezing distances and galloping distances are cut in half. These horses are usually coming from a classical training program where bucked shins is a common occurrence. When presented with these horses (often from a 2-yr-old in training sale), one is far better off putting them into this bone remodeling program initially rather than going on with them and hoping that they do not buck their shins, as they invariably do, and then take far longer to get to their first race. Sore horses should be individually evaluated based on their clinical and radiographic presentation. Most horses that present with the beginning sore shin can modify their training program and go on training. A short rest period with hand walking, phenylbutazone, and ice water can get most animals back into training rather quickly.

Although bucked shins are commonly accepted by veterinarians, trainers, and owners as a normal training event in young Thoroughbred racehorses, with estimated losses to the industry of only $10,000,000/yr in lost training and racing days, it is far more important than that! Approximately 12% of horses that buck their shins go on to develop

stress or saucer fractures later in their career. Besides the aggravation of yet another lameness occurring at the peak potential earning period in the horse's career, these are the animals at risk for mid-cannon bone fractures, which represent approximately 10% of the fatal catastrophic musculoskeletal breakdown injuries that occur on the racetracks in North America each year.

6. Conclusions

Although conclusions have been drawn within each experiment described in this paper, there are several general conclusions that can be drawn from the experience of studying this condition for more than 20 years. We now have a better understanding of the mechanical and biological events that lead to bucked shins.

1) It seems that horses are not born with the right bone structure for racing. They must develop it. Bone can only develop based on its own experience (Wolff's Law). Training adapts bone to training and training that mimics racing adapts bone to racing.
2) It is important to change the inertial properties of MCIII to resist bending in the dorsopalmar direction.
3) Different exercise programs can change the shape and substance of bone (modeling and remodeling) with hard surfaces giving faster results.
4) High-speed exercise in small doses seems highly protective against bucked shins, whereas long galloping exercise increases the risk for bucked shins.
5) Exercise programs exist to decrease the incidence of bucked shins.
6) Horses that do not develop bucked shins do not develop stress or saucer fractures in their dorsolateral cortex.
7) Horses that do not develop stress or saucer fractures do not develop midshaft MCIII fractures while training and racing.
8) Institution of an effective training program for young Thoroughbreds to significantly decrease the incidence of bucked shins will therefore significantly decrease the incidence of fatal musculoskeletal mid-cannon bone fractures during racing and training.

Finally, it has been a wonderful experience to undertake/participate in this effort to understand bucked shins and to see the pieces fall into place over the years. With the completion of the recent prospective and retrospective study with Ray Boston, the "wagons have been circled": the results of these research efforts have clinical relevance for the Thoroughbred racehorse and the racing industry.[13] Bucked shins do not have to be a part of normal training for racing of young Thoroughbred racehorses. Although the way is clear for the Thoroughbred racehorse, there are still some questions this wonderful, naturally occurring model of fatigue failure can answer to improve our basic understanding of fatigue in bone.

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