DENSITY AND GEOMETRY OF THE THIRD METACARPAL IN JUVENILE RACEHORSES TREATED WITH EXOGENOUS EQUINE SOMATOTROPIN

A Dissertation

by

KATHERINE LENORE THOMSON

Submitted to the Office of Graduate Studies of
Texas A&M University
in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

August 2005

Major Subject: Animal Science

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Co-Chairs of Committee,	Gary D. Potter
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ABSTRACT

Density and Geometry of the Third Metacarpal in Juvenile Racehorses
Treated with Exogenous Equine Somatotropin. (August 2005)
Katherine Lenore Thomson, B.S.; M.S., Tarleton State University;
D.V.M., Texas A&M University
Co-Chairs of Advisory Committee: Dr. Gary D. Potter
Dr. Pete G. Gibbs

The effect of exogenous somatotropin (eST) on bone changes were evaluated in twenty-nine juvenile horses in race training using radiographs of the third metacarpal obtained over the course of a 128 day research project. A biodensitometer was used to measure bone density, and a micrometer was used to measure cortical bone width and medullary cavity width. Fifteen horses were given daily intramuscular injections of eST and fourteen horses were given daily intramuscular injections of sterile saline and served as the control group.

By day 128, the increase in total radiographic bone aluminum equivalence (RBAE) was significantly greater in the eST horses than in the control horses. The increases in RBAE in the dorsal and the medial cortices were greater in the eST horses than in the control horses, but these differences were not significant. There was a trend for changes in the ratio of RBAE in the dorsal to palmar and in the medial to lateral cortices to be greater in the eST than in the control horses.

By day 128, the increases in both the dorsal and the medial cortical bone width were significantly greater in the eST than in the control group of horses. The eST horses had a significantly greater decrease in dorsal to palmar medullary cavity width, and increase in dorsal to palmar bone diameter than the control group. A computed index of dorsal cortical bone increased significantly more in the eST than in the control group.

The stresses applied to bone are greater in the dorso-medial direction in racehorses. To decrease the strain, bone must either increase in bone mineral density, cortical width, and/or bone diameter. Both the eST group and the control group did make these changes

in bone over time, but the eST group more effectively remodeled and modeled bone to increase the strength of the third metacarpal than did the control group of horses.

In this research project, exogenous somatotropin treatment had a positive effect on the density and geometry of the third metacarpal. These changes are believed to result in a decreased risk of bone injury to the eST treated horses.

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TABLE OF CONTENTS

Page

Page

Page

LIST OF FIGURES

FIGURE Page

FIGURE Page

FIGURE Page

FIGURE Page

FIGURE Page

FIGURE Page

LIST OF TABLES

TABLE Page

TABLE Page

TABLE Page

TABLE Page

TABLE Page

TABLE Page

INTRODUCTION

Bone is a dynamic tissue that adjusts to the stresses placed upon it. Long bone that is stressed beyond its physiological limit normally undergoes a response that includes enlarging its periosteal diameter to reduce the strain placed on the bone. Also, in bone that has experienced microfractures, the damaged areas of bone normally undergo resorption by osteoclasts and then new bone material is laid down by osteoblasts. Both of these methods, modeling and remodeling, allow bone to adjust to stress placed upon it. However this adjustment takes time. If bone is not given enough time to adjust, serious injury is more likely.

Juvenile racehorses are often exposed to the amounts of repetitive stress that can cause stress fractures. Allowing these juvenile horses more time to mature might decrease the likelihood of injury. However, the current economics of the racing industry encourages racing of young horses. As a result, researchers have been looking at other ways to decrease the prevalence of bone injuries in horses.

Stress fractures and microfractures can be caused by stress beyond the normal physiological limit, stress within the normal physiological limit that is applied in a different plane than normal, or physiologically normal amounts of stress if the stress is repeated often enough. One way to reduce the likelihood of stress fractures is by causing positive changes in bone modeling and/or remodeling. Increasing the mineral density of a bone and/or increasing the diameter of a bone should result in decreased strain placed upon that bone by any given amount of stress. In direct contrast to this ideal, juvenile racehorses typically undergo a decrease in bone mineral density of the third metacarpal that often occurs near the time that trainers increase the speed demands on these horses. Speed is thought to increase the strain placed upon these bones, thereby increasing the likelihood of the development of stress fractures and/or microfractures. Other bones may also undergo a decrease in bone mineral density due to remodeling as a result of training.

This dissertation follows the style and format of the Journal of Animal Science.

Bone modeling and remodeling are both influenced by specific hormones. Growth hormone, also known as somatotropin, has been shown to increase production of insulin-like growth factor 1 (IGF-1) and osteoblastic activity. Increased osteoblastic activity results in increased bone deposition.

One technique to influence bone deposition positively in juvenile racehorses may be the administration of exogenous somatotropin to increase circulating IGF-1, thereby increasing osteoblastic activity and resulting in an increase in bone mineral density and/or bone diameter. Either of these results could decrease the likelihood of injury to juvenile racehorses.

REVIEW OF LITERATURE

The most important cause of training failure in Thoroughbreds is lameness (Lidner and Dingerkus, 1993). Pre-existing stress fractures are often seen in humeral (Johnson, 1993; Johnson et al., 1994; Stover et al., 1991), pelvic (Stover et al., 1993; Johnson et al., 1994), scapular (Stover et al., 1993) and tibial (Stover et al., 1993) fractures. Musculoskeletal injuries account for the majority of racing and training deaths in both Thoroughbreds and Quarter Horses (Johnson, 1993; Estberg et al., 1993, 1996). Musculoskeletal injuries in racehorses are a problem in the United States, Europe, and worldwide.

Prevalence of Racehorse Injuries

Racehorses in Australia have an incidence rate of 2.4 (Bailey et al., 1997) and 1.1 (Bourke, 1994) musculoskeletal breakdowns and fatalities, respectively, per 1000 starts. Assuming 10 horses per race, 10 races per day, and 5 racing days per week, these indicate rates would be 1.2 breakdowns and 0.55 fatalities per week at a race track. In Southern Africa 0.14% of starters broke down during racing (Macdonald and Toms, 1994).

At Cologne, Germany, 57% of training failures were due to lameness (Lindner and Dingerkus, 1993).

A study in Newmarket found racehorses had a 9% annual incidence of fracturing a bone (Bathe, 1994). Of the 245 fractures observed, 11 were dorsal metacarpal stress fractures. This study found that the majority of fractures occurred during training rather than racing, as have other studies (Kobluk et al., 1992; Wilson et al., 1996; Verheyen and Wood, 2004) while some studies found Thoroughbred fatal injuries were equally associated with racing and training (Johnson, 1993; Estberg et al., 1993; Johnson et al., 1994). The majority of Quarter Horse fatal injuries occurred during racing (Johnson, 1993; Johnson et al., 1994).

British Thoroughbred racehorses suffered 1.15 fractures per 100 horse months (Verheyen and Wood, 2004). Analysis of the 148 fractures included in the study found that 22 were stress fractures of the third metacarpal.

During 1992, in the United States, 0.32% of Thoroughbred 2-year-old starters on dirt tracks suffered an injury while only one injury was reported on a turf track (Wilson et al., 1996). Five of the injuries on the dirt tracks were bucked shins and another 7 of the total 57 reported were fractures of the third metacarpal. Fatal injuries occurred in 0.159% of overall starts.

In California, 0.17 per 100 race entrants sustained a fatal musculoskeletal injury (Estberg et al., 1996). This is about 1 fatality every 6 racing days. Of the 79 fatal musculoskeletal injuries they observed during racing, 19 involved fracture of the metacarpus. When they also included horses that sustained an exercise-related fatal injury during training, they found 41 of the 163 injuries involved third metacarpal fractures (Esterberg et al., 1993). This study was then extended for 3 more years and found that in Thoroughbred horses, 75 of 258 fatal fractures during racing and 61 of 286 fatal fractures during training involved the third metacarpal bone (Johnson et al., 1994). These same authors noted 2 of 24 fatal musculoskeletal injuries in Quarter Horses during one year were fractures of the third metacarpal (Johnson, 1993).

Haynes and Robinson (1988) studied Thoroughbreds at Canterbury Downs and observed that 81% developed musculoskeletal injuries. Additionally they reported that 35% of the horses sustained musculoskeletal injuries that were severe enough to prevent training or racing. These authors noted 3.2 breakdowns per 100 horses that started in at least one race (Haynes and Robinson, 1988; Robinson et al., 1988).

A study in New York concluded that the fracture incidence during races for Thoroughbreds in that state was approximately 2 per 100 horses (Hill et al., 1986). Metacarpal bone fractures accounted for 13% of the musculoskeletal injuries observed (Mohammed et al., 1991).

Thoroughbred racehorses in Kentucky suffered injuries at a rate of 0.33% per start and catastrophic injuries at a rate of 0.14% per start (Peloso et al., 1994).

The exact percentages of injuries to racehorses vary in these reports. Besides normal chance variation in the amount of injuries, different authors used different criteria to determine what constituted an injury and used different types of animals for their control group. Additionally, the reports from outside the United States were from areas that tend to run the majority of their races on turf rather than on dirt tracks, which has been speculated to affect the rate of injury (Mohammed et al., 1991). Even with these variations these reports verify that musculoskeletal injuries are of great concern to the horse racing industry.

Factors Associated with Racehorse Injuries

Authors have looked at various factors that may influence racetrack injuries, but have been unable to agree on all the causative agents.

They have found that increasing age does not (Hill et al., 1986), or alternately does (Haynes and Robinson, 1988; Robinson et al., 1988; Mohammed et al., 1991; Kobluk et al., 1992; Bourke, 1994; Macdonald and Toms, 1994; Estberg et al., 1996; Bailey et al., 1997) increase a horse's risk of injury. Verheyen and Wood (2004) found that young horses suffered more fractures during training, while aged horses were more likely to experience a fracture during racing than during training. Fillies have more (Lidner and Dingerkus, 1993), or alternately fewer injuries than males (Macdonald and Toms, 1994; Estberg et al., 1996; Wilson et al., 1996). Differences in breeds have been noted, but are confounded by the different types of racing associated with the various breeds (Nunamaker, 1986; Nunamaker et al., 1990; Pool and Meagher, 1990; Wilson et al., 1996).

Track conditions have been contemplated as contributing to racehorse injuries. Dirt tracks have been observed to be associated with an increased risk of injury over turf tracks (Mohammed et al., 1991), while no significant difference between the type of track was seen by others (Hill et al., 1986; Robinson et al., 1988; Kobluk et al., 1992).

Kane et al. (1996) investigated the type of horseshoe as being a contributing factor to injury. They found that the likelihood of injury was increased by the use of front toe

grabs and decreased when horses were shod with rim shoes. Pratt (1997) also noted that toe grabs increase the rate of injury.

Some authors have documented a negative correlation between risk of injury and the cumulative exercise distance during the time period preceding a race (Kobluk et al., 1992; Cohen et al., 2000) while other authors noted a positive correlation (Lidner and Dingerkus, 1993; Estberg et al., 1994, 1995, 1998). Robinson and others (1988) saw a significant difference in injury occurrence between trainers and theorized that it was due to differences in the exercise programs used by the various trainers. Haynes and Robinson (1988) found an increased risk for horses that had raced within the previous 12 days, but a decreased risk for animals with higher overall exercise scores. In this study they also observed that horses with a catastrophic injury often had a preceding injury.

Horses with more starts per year tend to have fewer breakdowns (Haynes and Robinson, 1988; Mohammed et al., 1991; Kobluk et al., 1992). Horses in stakes races (Bailey et al., 1997) or horses in claiming races (Haynes and Robinson, 1988; Robinson et al., 1988; Kobluk et al., 1992) were found to have an increased chance of injury in some studies, while no difference was seen in another study (Wilson et al., 1996).

Other factors that have been contemplated as influencing the injury rate to racehorses include field size (Bailey et al., 1997), barrier position (Bailey et al., 1997), distance ran in the last preceding race (Bailey et al., 1997), location on the track (Hill et al., 1986; Robinson et al., 1988; Kobluk et al., 1992; Mohammed et al., 1992; Bourke, 1994; Wilson et al., 1996), distance of the race (Hill et al., 1986; Kobluk et al., 1992; Bourke, 1994; Peloso et al., 1994), hoof angle (Kobluk et al., 1992), conformation (Kobluk et al., 1992), season of the year (Mohammed et al., 1991), season of racing (Mohammed et al., 1991), and number of days between previous races (Peloso et al., 1994).

Stress Fractures, a Specific Type of Injury

Stress fractures are bone fractures, either partial or complete, that are not associated with a single traumatic episode (Carter and Hayes, 1977). Instead they are caused by repetitive occurrences of stress that are beneath the fracture threshold (Burr, 1997). They

are one of the most commonly occurring overuse injuries experienced by athletes (Jones et al., 1989). Stress fractures are well documented in military recruits and have been discussed by various authors (Scully and Besterman, 1982; Burr, 1997; Popovich et al., 2000). Some authors reserve the term stress fracture for those cases with a fracture line visible by radiograph (Stover et al., 1988). Fatigue fracture is another term that is often used to refer to this type of bone damage.

One type of stress related injury that often occurs in racehorses is dorsal metacarpal disease, also commonly called "bucked shins". In dorsal metacarpal disease microfracture of the cortical bone is thought to be the primary lesion (Stover et al., 1988). Clinical signs of dorsal metacarpal disease include heat, swelling, and pain upon palpation of the dorsal metacarpal area while necropsy signs are congestion and edema of the periosteum and subcutaneous tissue associated with the dorsal metacarpal area (Katayama et al., 2001). Dorsal metacarpal disease has been found to occur in 42% (Bailey et al., 1999), or 59% (in one study) and 70-80% on average (Larkin and Davies, 1996) of 2-year-old Thoroughbred racing horses in Australia. It occurs in 70% (Norwood, 1978) to 91% (Stover et al., 1988) of 2-year-old Thoroughbred racing horses in the United States, and in 5 - 50% of racing Quarter Horses (Goodman, 1987). Shin soreness was found to be the most common injury occurring to Thoroughbred 2-year-olds in race training in Australia (Bailey et al., 1999). In a study in Australia, they found that of the horses that developed dorsal metacarpal disease as two-year-olds, 40% had a recurrence sometime during their two or three year-old years (Bailey et al., 1999). In contrast, Nunamaker et al. (1990) state that once a horse has recovered from bucked shins the condition rarely recurs.

Another stress related injury that occurs in racehorses is tibial stress fracture (Bathe, 1994, Verheyen and Wood, 2004). Additionally, humeral fractures in racehorses often show signs of pre-existing stress fractures (Stover et al., 1991; Johnson, 1993).

Various authors have reported that long bone fractures of racehorses often show evidence of a pre-existing stress fracture (Stover et al., 1991; Johnson, 1993; Stover et al., 1993; Johnson et al., 1994) or other pre-existing pathological condition (Pool and

Meager, 1990; Kobluk et al., 1992; Stover et al., 1994). Thus many long bone fractures in racehorses may begin with microfracture damage to the cortical bone.

Microfractures in Cortical Bone

Cortical bone is the dense compact bone that makes up the shafts of long bones and the outer layer of all bones. Microfractures are typically defined as a matrix failure that can be detected by light microscopy (Burr et al., 1997). Microfractures have been seen to occur in cortical bone loaded *in vivo* (Mori and Burr, 1993; Burr et al., 1997) and *in vitro* (Carter and Hayes, 1977). Horses in race training have microfractures (Stover et al., 1992). Microfractures weaken the bone and may therefore lead to catastrophic failure (Schaffler et al., 1989). Tension damage to bone resulting in modulus reduction of less than 20% does not significantly reduce the impact strength of the bone (Reilly and Currey, 2000). In contrast, bone damaged in compression, and then loaded in tension appeared to have a reduction in impact strength that presumably results from the long and discrete microcracks produced by compression (Reilly and Currey, 2000). However, if bone remodeling can repair the microfracture damage before it becomes too great, bone failure may be delayed or prevented (Martin and Burr, 1982).

Intracortical Bone Remodeling

Intracortical bone is remodeled by a several step process. The steps are coupled to one another and always proceed in the same order with bone resorption preceding bone deposition (Parfitt and Chir, 1987; Pool, 1991). The remodeling is done in individual packets of bone cells referred to as basic multicellular units (BMU) (Frost, 1987).

The first step is the least understood. Somehow the bone reabsorbing cells, osteoclasts, are activated and begin resorbing bone at a specific site. This activation has been postulated to be triggered by strain magnitudes (Lanyon, 1984; Frost, 1987, 1990; Burr et al., 1989) that may be sensed by osteocytes, the resident cells of bone (Lean et al., 1996), stress-related electrical potentials (Davidovitch et al., 1984; Lanyon, 1993),

microfractures (Carter, 1984; Frost, 1990; Lanyon, 1993; Mori and Burr, 1993), retraction of bone lining cells (Mundy, 1990), fluid shear stress as sensed by osteocytes (Johnson, 1984; Lanyon, 1993; Burger et al., 1995; Parfitt et al., 1996; Sakai et al., 1999). and/or the loss of osteocyte integrity as a result of microfractures (Vashishth et al., 2000; Verborgt et al., 2000). Recently Yasuda et al. (1999) reported the discovery of an osteoclastic differentiation factor which can be expressed by cells of the osteoblastic lineage and appears to be a major ligand involved in mediating osteoclast activation. This may be related to osteoprotegerin, a glycoprotein found by Simonet et al. (1997), that also inhibits osteoclastic activation. Once activated, mononuclear precursor cells from the hematopoitic cell line combine to form the large, multinucleated osteoclasts (Mundy, 1990; Athanasou, 1996).

The second step is resorption. Resorption takes place by individual osteoclast cells forming a ruffled border with the bone (Athanasou, 1996), sealing these edges to the bone and using proton pumps to pump proteolytic enzymes and hydrogen ions into this "clear zone" (Mundy, 1990). Once an osteoclast has resorbed a given area of bone, it can become motile and move to a new area of bone (Mundy, 1990). Teams of osteoclasts erode a cutting cone in the cortical bone at a rate of 20-40µm (Parfitt and Chir, 1987) to 50µm (Jaworski, 1984) per day in a direction that is roughly parallel to the long axis of the bone. The rate and duration of bone resorption are thought to be regulated by genetics, local and systemic factors (Jaworski, 1984), with some of these factors working indirectly through osteoblasts (Athanasou, 1996). According to Parfitt and associates (1996), throughout the life of the BMU new osteoclast precursors are recruited, this recruitment is done by the osteoclasts, and this determines the rate and duration of the cutting cone. The precise signal for this recruitment is speculated to involve interleukin-6, annexin-II, and /or interleukin-1 (Mundy, 1990; Athanasou, 1996; Parfitt et al., 1996; Qu et al., 1999; Franchimont et al., 2000). The resorption stage takes 1 to 3 weeks (Parfitt and Chir, 1987).

Next there is a reversal phase that lasts for 1 to 2 weeks (Parfitt and Chir, 1987) at any given location of the cutting cone. During this step osteoblasts, the bone forming cells, are recruited to the cutting cone (Jaworski, 1984; Parfitt and Chir, 1987). Osteoblasts are

cuboidal and originate from the mesenchymal stem cells (Baron, 1990; Rodan, 1992). Analogously to the osteoclasts, osteoblasts are recruited as pre-osteoblast cells and the precise mechanism of signaling involved in recruitment is not known but may be strain-regulated (Smit and Burger, 2000). Once at the site of the cutting cone, the pre-osteoblasts differentiate into mature osteoblasts (Rodan, 1992). Unlike osteoclasts, osteoblasts are not motile and stay at the same site as long as they are laying down bone (Jaworski, 1984). However, as the osteoclasts continue to advance the front of the cutting cone, more osteoblasts are recruited to the newly resorbed areas of the cutting cone (Jaworski, 1984).

The fourth step is bone formation. This is a two-part process. First the teams of osteoblasts (100-400 per team) (Baron, 1990) produce and secrete the protein matrix of bone, which is 85-90% type 1 collagen (Baron, 1990; Teitelbaum, 1990; Termine, 1990; Rodan, 1992). The matrix is secreted at the approximate rate of 0.5µm (Rodan, 1992), 1.0-1.5µm (Jaworski, 1984), or 2-3µm per day (Parfitt and Chir, 1987). As they deposit the matrix some osteoblasts become embedded in the matrix and then change to become osteocytes (Jaworski, 1984; Rodan, 1992). The collagen matrix must undergo maturation for 5-10 days before the second part of the bone formation process can take place (Parfitt and Chir, 1987; Baron, 1990; Puzas, 1990). The collagen matrix that lies between the osteoblasts and the mineralized bone surface is called the osteoid seam and is 5-50µm thick (Puzas, 1990). Osteoblasts control matrix maturation and the subsequent mineralization of the bone (Puzas, 1990). Most of the mineral in bone is in the form of spindle-shaped crystals of hydroxyapatite, $Ca_{10}(PO_4)_6(OH)_2$ (Baron, 1990; Puzas, 1990). Because new osteoblasts are recruited as long as the osteoclasts are resorbing bone, the mineralization front follows the osteoid front, which follows the resorption front (Jaworski, 1984). During the first 2 weeks of mineralization about 70% of the mineral is deposited (Pool, 1991). The new bone does not reach its maximum density for 3-6 months (Parfitt and Chir, 1987).

The fifth step is quiescence. Remaining osteoblast cells flatten out and become bone-lining cells (Parfitt and Chir, 1987; Rodan, 1992) or disappear (Parfitt and Chir, 1987). The remodeling of this area of bone is complete.

Periosteal and Endosteal Bone Remodeling and Modeling

Periosteal and endosteal bone remodels in much the same manner as intracortical bone. The only real difference being that rather than a cutting cone, the BMU is in the form of a more diffuse (Jaworski, 1984) resorption pit, a Howship lacuna (Buckwalter et al., 1995).

However, in addition to remodeling, modeling can also take place on the periosteal and endosteal surfaces. Modeling differs from cortical remodeling in two important ways. First, modeling can involve the laying down of new bone without a preceding resorption phase (Pead et al., 1988; Baron, 1990; Bloomfield, 1995). Secondly, since bone resorption and formation are uncoupled, modeling changes the geometry of the bone (Baron, 1990; Teitelbaum, 1990). This change in geometry allows the bone to adapt to different stresses placed on it by modifying the cross-sectional area and/or changing the moment of inertia of the bone.

During growth, modeling is used to sculpt the metaphyses to retain the shape of the bone (Baron, 1990; Buckwalter et al., 1995). After the bone has reached its maximum length, modeling typically focuses on increasing the diameter of the bone by adding bone to the periosteal surface (Buckwalter et al., 1995) by intramembranous ossification (Pool, 1991). Modeling also typically decreases the thickness of the cortical bone by removing bone from the endosteal surface (Pool, 1991; Buckwalter et al., 1995). However, in foals, modeling normally increases the thickness of the dorsal cortex, as periosteal bone growth exceeds endosteal absorption in this area (Pool, 1991). In the young Thoroughbred racehorse modeling can add 1-2µm/day of lamellar bone to the periosteal surface (Nunamaker and Provost, 1991).

Effects of Exercise on Bone Modeling and Remodeling

According to Wolff's law, bones adapt to the stresses placed upon them. Bones that are overloaded, such as those in a young growing animal, are adapted by modeling (Jones et al., 1977; Goodship et al., 1979; Woo et al., 1981; Bloomfield, 1995). Even in

mature animals bone can adapt to increased strain by modeling (Lanyon, 1984; Meade et al., 1984; Rubin and Lanyon, 1984, 1985; Burr et al., 1989; Bloomfield, 1995). In both these cases, modeling increases the deposition of bone on the periosteal surface (Goodship et al., 1979) and may also decrease the resorption of bone from the endosteal surface (Jones et al., 1977; Woo et al., 1981). As noted previously this change takes time. New bone laid down in response to increased load was still not as dense or strong as mature bone 2 months after the increased strain was applied (Meade et al., 1984).

Bones that are underloaded, such as occurs in space flight (Cavolina et al., 1997), bed rest (Leblanc et al., 1990), disuse (Lanyon, 1984; Rubin and Lanyon, 1984, 1985; Skerry and Lanyon, 1995; Thomas et al., 1996), or deconditioning (Porr et al., 1998) are remodeled to decrease the amount of bone present.

Changing the shape of a bone, especially by increasing the outer diameter of the cortex, can decrease the strain experienced by that bone at a given level of stress. As the outside wall of the cortex of bone increases in diameter it, like any other cylinder, increases the area moment of inertia. In military recruits the area moment of inertia of the tibia correlates with the risk of stress fractures (Milgrom et al., 1989). Also, width of the tibia was significantly related to risk of stress fracture although cortical thickness was not (Giladi et al., 1987).

The third metacarpal (MC III) of a yearling Thoroughbred has a nearly round cross-section and a centrally located medullary cavity. The same cross-sectional view of an older Thoroughbred's third metacarpal shows a bone that has been modeled due to the strains placed upon it. The total area of cortical bone is increased and the area of the medullary cavity is also increased (Welch, 1999). The change in the shape of the bone is influenced by the exercise the horse undergoes (Welch, 1999).

In one study exercise was shown to increase bone modeling but decrease intracortical bone remodeling (Lanyon, 1984), though others have not seen changes in intracortical remodeling due to strain (Meade et al., 1984), or have seen an increase in intracortical remodeling (Goodship et al., 1979). Birch and Goodship (1999) saw a decrease in markers of bone remodeling in horses that were in high intensity training and suggest that this was due to exercise causing decreased remodeling. Frost (1990) theorized that

if enough microdamage occurs it will trigger cortical bone remodeling regardless of any normal depressive effect of exercise.

Juvenile racehorses have been shown to experience changes in bone mineral density of the third metacarpal that is related to exercise (McCarthy and Jeffcott, 1992; Nielsen et al., 1997; Porr et al., 1998, 2000).

Somatotropin

Somatotropin (STH) is also known as growth hormone. This hormone is a protein molecule made up of 191 amino acids in man (Guyton and Hall, 1996), and 190 amino acids in the horse (Ascacio-Martínez and Barrera-Saldaña, 1994). It is produced in the anterior pituitary by the somatotroph cells (Greenstein, 1994). Secretion of STH is regulated by the hypothalamus via the production of growth hormone releasing hormone (GHRH), which stimulates STH secretion and somatostatin, also from the hypothalamus, which inhibits the secretion of STH. Secretion of STH is increased by sleep, exercise, and stress (Reichlin, 1998). Release of STH is not steady, but instead is episodic, climbing during sleep, shortly after rising, and twice during the day (Greenstein, 1994), or up to 13 pulses per 24-hour period (Reilchlin, 1998). Feedback control is both a direct negative feedback loop, as increasing levels of STH cause a decrease in GHRH secretion, and an indirect negative feedback loop, as increasing STH causes an increase in circulating IGF-1 which causes a decrease in GHRH secretion (Reilchlin, 1998).

Exogenous STH has been used in humans and animals. In humans, STH has primarily been used to correct deficiencies in endogenous STH (Ohlsson et al., 1998). In animals, effects of exogenous STH have been studied since Asdell (1932) treated dairy goats with hypophyseal extract. In dairy cattle, STH treatment has been shown to increase the lactational yield (Bauman et al., 1999) and the amount of milk produced per amount of feed input (Bauman, 1992). In swine, exogenous STH has been shown to increase growth performance (Chung et al., 1985). Studies have found that STH does not (Veum et al., 1997), or alternately does (Wester et al., 1998; Wang et al., 1999) affect growth of neonatal pigs. In an overview of STH use in animal production, Etherton and

Bauman (1998) state that exogenous STH increases the food output per unit of feed input.

Unlike most hormones, STH does not target a specific organ but has its influence on almost all body tissues including bone (Guyton and Hall, 1996). Many of the effects of STH are indirect and occur due to STH stimulating the liver to increase its secretion of IGF-1.

Indirect Effects of Somatotropin on Bone Via IGF-1

Somatomedin, IGF-1, mediates many of the actions of STH (Greenstein, 1994). Under the influence of STH the liver increases its secretion of IGF-1 into the circulation. However the increase in serum IGF-1 does not occur until 12 hours after the rise in STH (Salih et al., 1999). In the circulation almost all IGF-1 is bound to IGF-1 binding proteins (IGFBP) (Rosen and Pollak, 1999). There are six known IGFBPs and they function to help control the rate at which IGF-1 is delivered to its target cells (Root, 1994). In bone it is primarily IGFBP-5 that allows storage of IGF-1 in the matrix tissue (Rosen and Pollak, 1999). In addition to increasing circulating levels of IGF-1, STH also stimulates bone to increase secretion of IGF-1 (Raisz et al., 1998) as well as other growth factors (Mohan and Baylink, 1990). As a result, STH causes an increase of IGF-1 at both systemic and local levels.

According to Canalis (1990), IGF-1 stimulates osteoblasts to replicate and to increase their production of bone protein matrix. In contrast, Puzas (1990) claims that osteoblasts do not replicate. While not affecting bone resorption, IGF-1 does decrease bone collagen breakdown (Canalis, 1990). Additionally, IGF-1 has anabolic effects on osteoblasts (Ohlsson et al. 1998).

Direct Effects of Somatotropin on Bone

Nilsson et al. (1995) established that osteoblasts have receptors for STH. Kassem and coworkers (1993) found that STH may have direct anabolic effects on osteoblasts, which

can stimulate osteoblast proliferation. Ohlsson et al. (1998) also noted that STH stimulates osteoblasts to proliferate. Thus, STH appears to affect bone metabolism in two ways, direct stimulatory effects on osteoblasts and indirect effects via increase in IGF-1 levels. Directly, STH may stimulate prechondrocytes and preostesoblasts, while indirectly, via IGF-1, STH stimulates more mature cells (Ohlsson et al., 1998).

Combined Use of Somatotropin and Exercise

A study by Oxlund and workers (1998) found that the combined use of STH and exercise increases cortical bone growth in rats. Similarly, Banu et al. (1999) found that STH and exercise had additive effects on some measurements of bone, as did Yeh and co-workers (1994).

A previous study in this laboratory found trends for exogenous STH to increase bone mineral density in juvenile racehorses (Julen-Day et al., 1998). Another study utilizing a larger group of horses was needed to further evaluate the potential beneficial effects of the combination of STH treatment and exercise in increasing bone strength in young racehorses.

MATERIALS AND METHODS

This was one of a two part study examining specific effects of exogenous somatotropin administration to juvenile horses in race training. The first part of this study has previously been published (Sutfin, 2000). This part of the study concentrated on changes in the third metacarpal that were measureable via radiographs.

Management of Animals

Thirty-two long-yearling Quarter Horses, each obtained from a volunteer horse owner across the United States as a loan for the duration of the project, were paired by age within sex (mean age at entry into study was 629 ± 11 days). One horse from each pair was randomly assigned to a treatment group and the other to a control group. During the study three horses were removed from the project. One of these horses had a pre-existing bone cyst, the other two horses developed extended lameness caused by stone bruises to the feet. A total of twenty-nine horses completed the project, fourteen in the control group and fifteen in the treatment group. Mean starting weight of the horses in the treatement group was 360.1 ± 13.3 kg, and 379.7 ± 10.2 kg in the control group.

The protocol for management of the animals was approved by the Texas A&M University Agriculture Animal Care and Use Committee. On arrival, all horses were vaccinated and dewormed using procedures and products typically prescribed for horses in Central Texas. Hoof care was provided as needed throughout the project. The horses were housed in 3.7m x 3.7m box stalls and exercised on a dirt training track. All of the horses were fed the same balanced diet formulated to supply 130% of the current National Research Council (1989) recommendations for protein, minerals and vitamins for horses in training. Complete composition of the ration was published previously (Sutfin, 2000).

The horses were fed the same diet throughout the study, with amounts offered per feeding adjusted to maintain a constant body condition score of 5 - 6 using the system

developed by Henneke et al. (1983). The study was conducted at Steephollow Farm, a racehorse training facility near College Station, Texas.

Treatments

Horses in the treatment (eST) group were given intramuscular injections of a recombinant equine growth hormone (EquiGen™)[1] daily at 1900h. The form of EquiGen™ used in this study was a powder that was reconstituted according to the manufacturer's directions. EquiGen™ contains 191 amino acids, one more amino acid than naturally occurring equine somatotropin. An extra methionine residue was intentionally added to facilitate growth in bacteria. The dosage administered was 10µg/Kg BW per day for the first 7 days, followed by 20µg/Kg BW per day for the remaining 121 days of the study, as per the manufacturer's recommendation. The horses in the control group were given intramuscular injections of an equivalent volume of sterile saline. All injections were given daily at 1900h. Injection sites alternated between the neck and the pectoral region.

The experiment was conducted in four, 32-day periods. On days 0, 32, 50, 64, 82, 96 and 128 of the study, radiographs were taken of the entire left third metacarpal with an aluminum (Al) stepwedge penetrometer attached to the radiographic cassette. After the initial radiographs were taken, the horses were started in training. The training regimen was similar to that reported by Nielsen et al. (1997) which was developed to be typical for Quarter Horse race training.

Training Protocol

During the first week the horses were worked six days and rested one day. The riding started on day one in a round pen and advanced to the track by day two or day three depending on the temperament of the horse. On the initial days, horses were walked 550

[1] BresaGen, Ltd., Adelaide, Australia

m, trotted 1100 m and galloped 275 m. For the rest of week one the horses were ridden the same as on day 2-3, except that an additional 275 m of galloping was added each day. By day six the horses were galloped 1375 m. In weeks two-to-four the horses were ridden five days per week. The horses were walked and trotted as in the first week and galloped 1650 m per day. The total distance galloped per week during these three weeks was 8250 m. During the next period of four weeks the horses were ridden four days per week. Total distance galloped per horse per week was increased to 8440 m. For the third period of four weeks the horses were again ridden four days per week. On three of these days the horses were trotted 550 m and galloped 1925 m, and on one day the horses were warmed up, sprinted, and galloped. The horses were sprinted 230 m and galloped a total of 8210 m each week during the third period. In the fourth period the program was the same as the third period except the sprint was increased to 275 m and the gallop was decreased to 8165 m per week. During the entire trial the horses were walked for at least one hour on a mechanical walker on non-riding days. At the start of the trial and during the last four days of each 32-day period the horses were confined to tie stalls for a total collection of feces and urine, which was used in another study.

Samples Collected for This Study

Dorsal-palmar and lateral-medial radiographic views of the left third metacarpal were taken with an Al stepwedge penetrometer attached to each radiographic cassette. The radiographs were taken at a focal distance of 28 inches, using 70 KVP, 20 mA and exposure times of 0.2 seconds (dorsal-palmar views) and 0.16 seconds (lateral-medial views)

Measurements of Bone Density

The radiographs were then scanned using a Bio-Rad Model 620 Video densitometer. A logarithmic regression was formed using the optical density (OD) of the steps of the Al penetrometer as a standard. The maximum OD of four cortices of the bone was

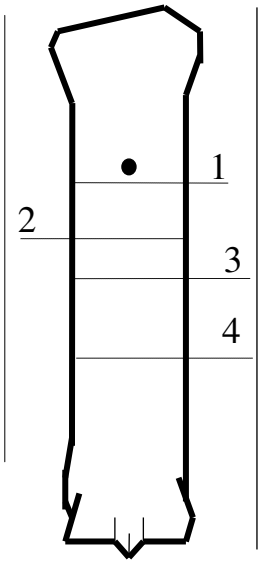
measured at a point 1 cm distal to the nutrient foramen and perpendicular to the long axis of the bone, and compared to the Al standard to determine bone density in radiographic bone aluminum equivalence (RBAE). This location on the bone was used both because the nutrient foramen serves as a landmark and because this location has been examined by several authors (Lawrence et al., 1994; Nielsen et al., 1997, 1998; Porr and Ott, 1997; Porr et al., 1998, 2000; Waite et al., 2000). This location is referred to in this paper as Site 1 (Figure 1).

Bone density was expressed as RBAE in millimeters of aluminum. Also, the area under the density curve from the dorsal-palmar radiographs was calculated and standardized with the corresponding area under the curve from the Al stepwedge on the corresponding radiograph to give an estimate of total bone density expressed as RBAE in mm^2Al.

It was possible that any changes in bone mineral density seen at the nutrient foramen would be unique to that location on the bone. To determine if these same patterns in bone mineral density occurred at other areas, additional locations on the third metacarpal were also examined using the same procedures as outlined above.

One area evaluated in this same manner was the mid-diaphysis region. Two different locations in this region were studied. One location was the midpoint of the bone as measured along the longitudinal axis from the carpal-metacarpal joint surface to the distal extreme of the articular condyles of metacarpal III. This area was chosen because it has been examined by several authors (Turner et al., 1975; Rybicki et al., 1977; Schryver, 1978; El Shorafa et al., 1979; Meakim et al., 1981; Nunamaker et al., 1989; Gross et al., 1992; McCarthy and Jeffcott, 1992; Gibson et al., 1995; Davies, 1996; Larkin and Davies, 1996; Skedros et al., 1996; Birch and Goodship, 1999) and also coincided with the area often affected by dorsal metacarpal disease (Stover et al., 1988; Nunamaker et al., 1990). This location is referred to in this paper as Site 2 (Figure 1).

The second location in this region was the midpoint of the bone as measured from the proximal end of the bone to the distal limit of the diaphysis as this area has been examined by Stover and coworkers (1992). This location is referred to in this paper as Site 3 (Figure 1).



- Site 1 1cm below nutrient foramen
- Site 2 Midpoint of bone from proximal end of bone to distal end of diaphysis
- Site 3 Midpoint of bone from proximal end of bone to distal end of articular surface
- Site 4 Junction of the proximal two-thirds and the distal one third of the bone

Figure 1. Diagram of the 4 sites of metacarpal III evaluated.

The final area to be evaluated in the same manner was the junction of the proximal two-thirds and the distal one third of the third metacarpal bone as measured along the longitudinal axis from the carpal-metacarpal joint surface to the distal extreme of the articular condyles of metacarpal III. This area was chosen because it is also often affected by dorsal metacarpal disease (Norwood, 1978; Stover et al., 1988) and is the area with the greatest cortical area in yearling Thoroughbreds (Nunamaker et al., 1989). This location is referred to in this paper as Site 4 (Figure 1).

It was anticipated that some of the radiographs would not show the entire third metacarpal. When this occurred, radiographs from the same horse, but on different dates were used to provide the needed total length in mm of the metacarpus. The specific locations to be evaluated were then measured from the nutrient foramen to ensure that the same location was measured on each successive radiograph of an individual horse. The equine third metacarpal only has one growth plate (Krook and Maylin, 1988), or at

least only one growth plate that is not closed before birth (Getty, 1975; Pool, 1991). This plate, located distally, closes at 6 - 12 months (Evans, 1990) or 6 - 18 months (Getty, 1975). Because the horses in this study were approximately 21 months old at the start of the study, the third metacarpal growth plates were closed and the length of the metacarpus of an individual horse would not vary over the course of the experiment. Radiograph evaluation confirmed the closure of the third metacarpal physes in these horses. Additionally, every effort was made to take true lateral-medial and dorsal-palmar views and to avoid radiographic aberrations by keeping the radiographic plates both parallel and as close as possible to the third metacarpal.

Measurements of Bone Geometry

Geometric changes of the third metacarpal bone were also evaluated on these radiographs at the same locations where bone mineral density was evaluated. Because it was anticipated that measurable changes in the geometry of the third metacarpal would occur slowly, only radiographs from day 0, day 64 and day 128 were used for geometric measurements. A digital caliper was used to measure the thickness of the dorsal, medial, lateral and palmar cortical bone and the width of the medullary cavity in a manner similar to that reported by Larkin and Davies (1996). A radiographic view box and magnifying lens was used to assist in determining the edges of the cortical bone. These data were then analyzed to determine if measurable changes in the geometry of the third metacarpal of the individual horses occurred over the course of this experiment. Because changes in geometry were seen, further analyses were run to see if the changes were consistent over the two treatment groups.

Larkin and Davies (1996) report a significant correlation between shin soreness in racehorses and an Index calculated by using the dorsal and palmar cortical bone widths and the medullary cavity width. The Index describes the extent to which bone is modeled for increase of dorsal cortical bone relative to palmar cortical bone. This Index was calculated on the horses in this experiment on day 0, day 64 and day 128 using the method of Larkin and Davies (1996).

Index = X * Y, where X = (T-M)/M, and Y = D/P
D = dorsal cortical bone width, M = medullary cavity width,
P = palmar cortical bone width, and T = D + M + P

As changes were seen in the Index values over the course of the experiment, analyses were run to determine if treatment with STH influenced the magnitude of these changes in the Index.

Statistical Analyses

SAS 8.1 was used to analyze the data and is the source of the statistics reported in this paper. To compare effects of treatment and time on measurable changes in bone, much of the data were normalized to day 0 values. Both measured values and normalized values are reported in this paper.

ANOVA Repeated Measures, type III, with day, treatment, and day*treatment as the effects, and the degrees of freedom determined by the Satterthwaite method were used to analyze the changes over time in RBAE in the dorsal, palmar, lateral and medial cortices, and total RBAE; changes over time in micrometer readings of the dorsal, palmar, lateral and medial cortical bone width, lateral-to-medial and dorsal-to-palmar medullary cavity width, and lateral-to-medial and dorsal-to-palmar bone diameter; changes over time in the ratios of dorsal/palmar, lateral/palmar, medial/palmar, and medial/lateral RBAE cortical measurements; and changes over time in the Index. Least square means were used to identify time by treatment interactions using Tukey's procedure to declare significant differences. Each of the four sites measured on the third metacarpal were evaluated independently. Significant difference was set at $P \leq 0.05$ throughout this study. Trends, when reported, were set at $P \leq 0.10$.

RESULTS

The daily injections of eST did not result in any observable side effects on the horses. The horses treated with eST had an average feed consumption of 7.8 kg/day while the controls consumed an average of 8.1 kg/day. The difference between groups in average daily feed consumption was small and not unexpected as the eST horses began the project with a slightly lower average body weight than the control horses.

Total RBAE

Site 1

Total RBAE at site 1 was significantly affected by day (P≤.0001) but not by treatment or day*treatment (Figure 2, Tables A-1 and B-1). The differences seen between the two treatment groups in total RBAE at Site 1 at the start of the project had disappeared by the end of the trial.

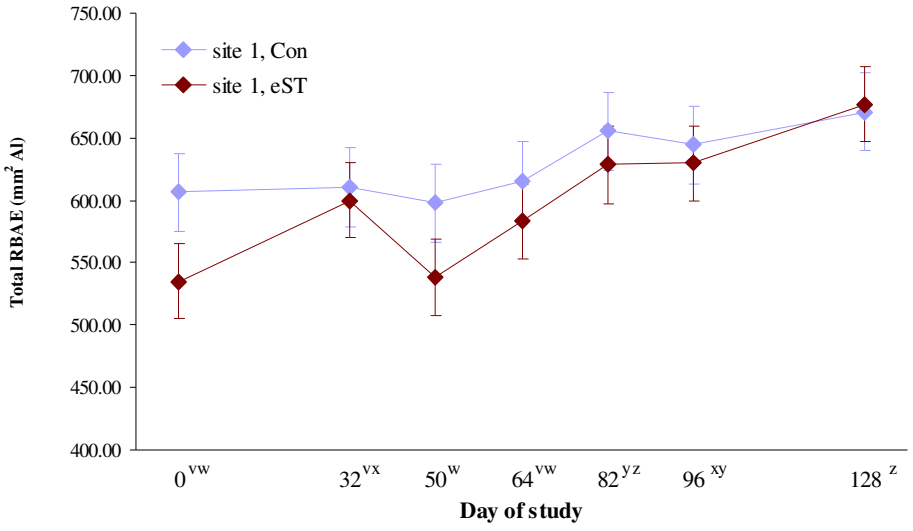


Figure 2. Total radiographic bone aluminum equivalence (RBAE) (mm^2 Al) at site 1. ^{vwxyz} Days not sharing the same superscript differ (P≤.05).

Data were normalized to remove the differences that were present between treatment groups on day 0 (Figure 3, Tables A-2 and B-2).

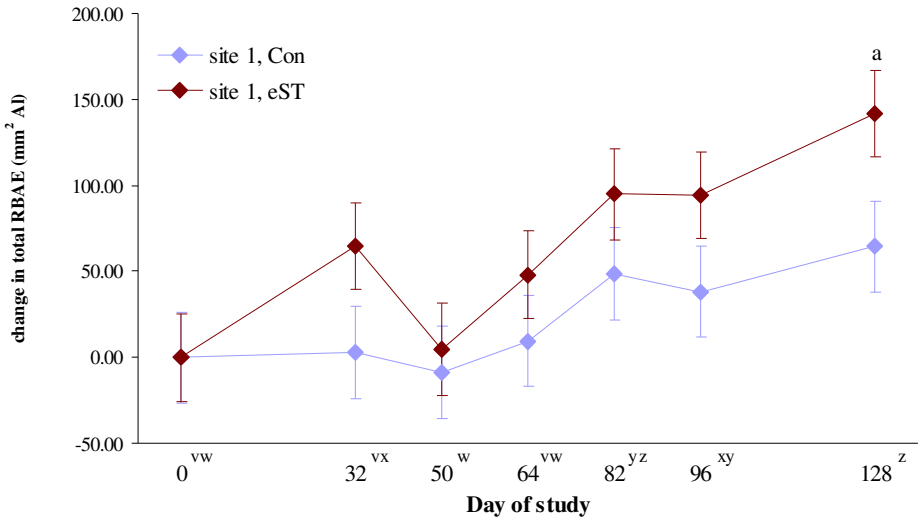


Figure 3. Normalized total radiographic bone aluminum equivalence (RBAE) (mm^2 Al) at site 1.
[a] Treatments differ (P≤.05).
[vwxyz] Days not sharing the same superscript differ (P≤.05).

Both treatment groups showed similar patterns of gain in total RBAE with an increase in total RBAE from day 0 to day 32, decline in total RBAE from day 32 to day 50, increase in total RBAE from day 50 to day 82, decline in total RBAE from day 82 to day 96, and a final gain in total RBAE from day 96 to the end of the study. The main difference seen between the groups was a greater magnitude of gain in total RBAE in the eST treatment group that resulted in a significantly greater gain in total RBAE by day 128 than that seen in the control group (P<.05).

Site 2

Total RBAE at site 2 was significantly affected by time (P<.0001) but not by treatment or day*treatment (Figure 4, Tables A-1 and B-3).

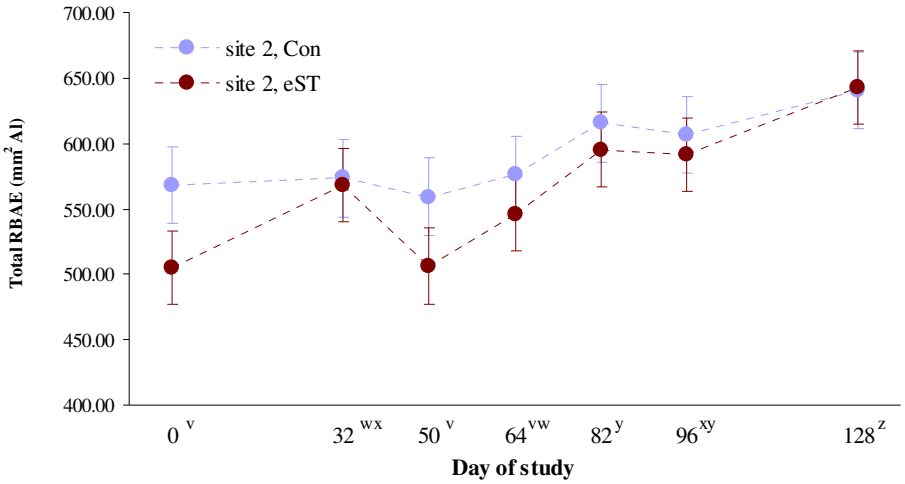


Figure 4. Total radiographic bone aluminum equivalence (RBAE) (mm^2 Al) at site 2. ^{vwxyz} Days not sharing the same superscript differ (P≤.05).

Differences that were seen between the two treatment groups on day 0 had disappeared by day 128. Data were normalized to day 0 values to better view any differences between the treatment groups in the change in total RBAE over time (Figure 5, Tables A-2 and B-4).

The overall pattern of gain in total RBAE at site 2 was very similar to that seen at site 1 (Figure 3), with a general gain in total RBAE over the 128 days of the trial in both groups, but a decrease in net gain seen on days 50 and 96. There was a trend for the two treatment groups to differ on day 128, with the eST horses having a greater gain in total RBAE than the control horses (P=.07).

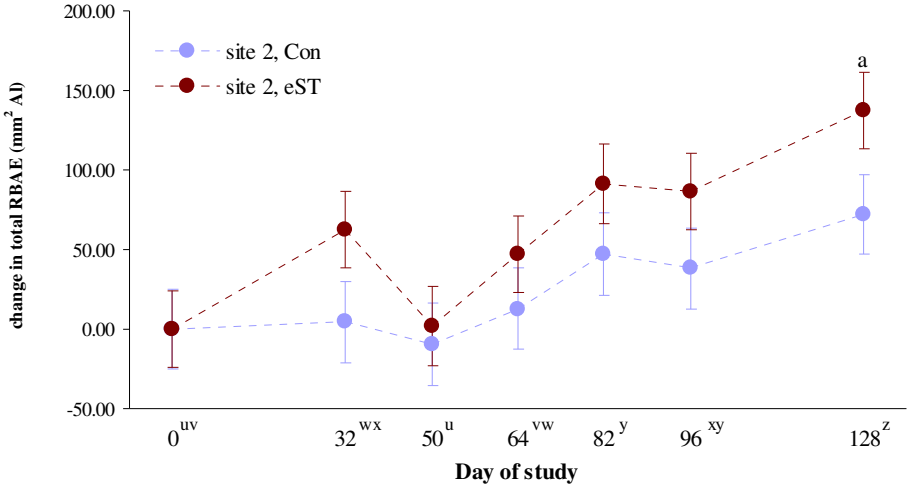


Figure 5. Normalized total radiographic bone aluminum equivalence (RBAE) (mm^2 Al) at site 2.
[a] Trend for treatments to differ (P≤.10).
[uvwxyz] Days not sharing the same superscript differ (P≤.05).

Site 3

Total RBAE at site 3 was significantly affected by time (P<.0001) but not by treatment or day*treatment (Figure 6, Tables A-1 and B-5).

Differences that were seen between the two treatment groups on day 0 had disappeared by day 128. Data were normalized to day 0 values to better view any differences between the treatment groups in the change in total RBAE over time (Figure 7, Tables A-2 and B-6).

Similar to the changes in total RBAE seen at site 1 (Figure 3) and site 2 (Figure 5), the overall pattern of net increase in total RBAE at site 3 was interrupted by a decrease in total RBAE on days 50 and 96. The eST horses had a trend for greater net increase in total RBAE at site 3 on day 32 (P=0.09) and on day 128 (P=0.07) than the control horses.

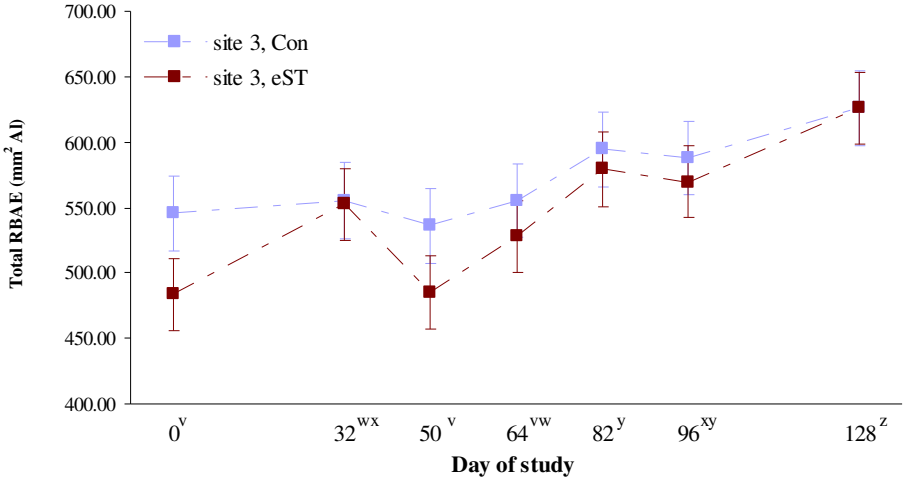


Figure 6. Total radiographic bone aluminum equivalence (RBAE) (mm^2 Al) at site 3.
^{vwxyz} Days not sharing the same superscript differ (P≤.05).

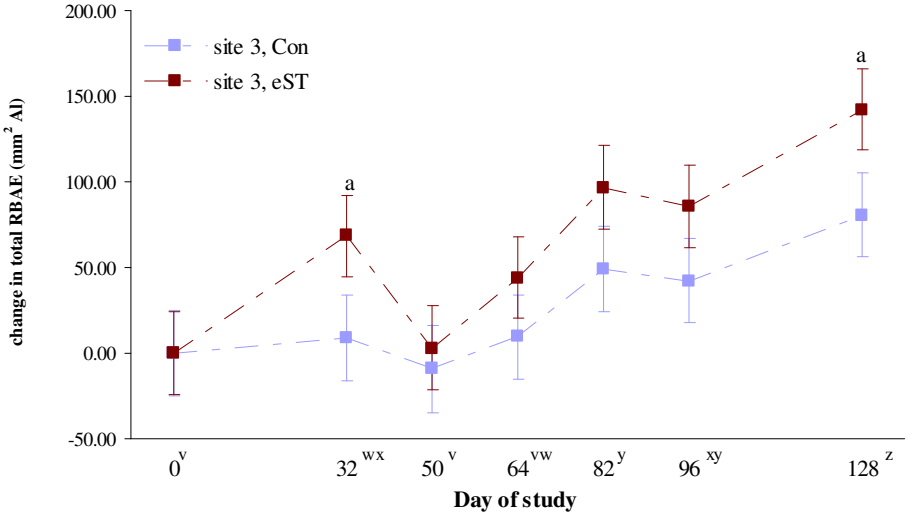


Figure 7. Normalized total radiographic bone aluminum equivalence (RBAE) (mm^2 Al) at site 3.
^{a} Trend for treatments to differ (P≤.10).
^{vwxyz} Days not sharing the same superscript differ (P≤.05).

Site 4

Total RBAE at site 4 was significantly affected by time (P<.0001) but not by treatment or day*treatment (Figure 8, Tables A-1 and B-7). Once again, the differences in total RBAE that existed between the two treatment groups at the start of the project had disappeared by the end of the 128 days.

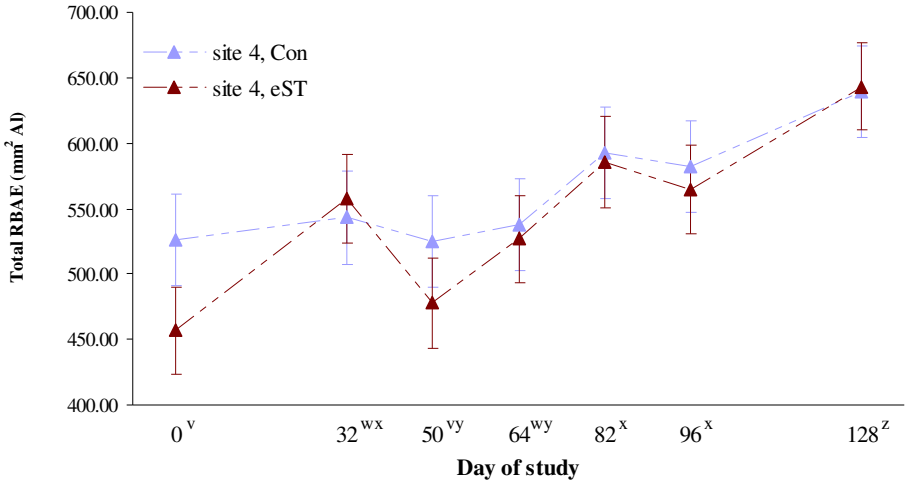


Figure 8. Total radiographic bone aluminum equivalence (RBAE) (mm^2 Al) at site 4. ^{vwxyz} Days not sharing the same superscript differ (P≤.05).

Data were normalized to day 0 to better view the changes in total RBAE over time (Figure 9, Tables A-2 and B-8). Both of the two treatment groups had a net gain in total RBAE at site 4 over the 128 day trial, with decreases in total RBAE measured on days 50 and 96. There was a trend for the eST group to have a greater gain in total RBAE than the control group at site 4 on day 32 (P=.07). On day 128 the difference in gain of total RBAE between the two treatment groups at site 4 was not significant (P=.101).

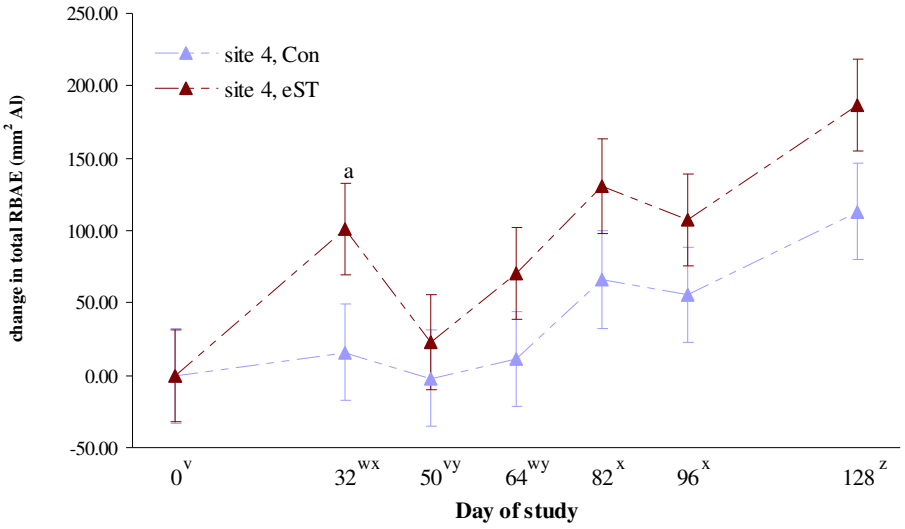


Figure 9. Normalized total radiographic bone aluminum equivalence (RBAE) (mm^2 Al) at site 4.
[a] Trend for treatments to differ (P≤.10).
[vwxyz] Days not sharing the same superscript differ (P≤.05).

Dorsal RBAE

Site 1

A significant day effect was seen in dorsal RBAE at site 1 (P<.0001), but there was not a treatment*day interaction (Figure 10, Tables A-3 and B-9). There was a trend for a difference (P=.09) between the two treatment groups, with the control horses having a greater amount of cortical bone in the dorsal cortex than the eST horses (as measured by RBAE) at the start of the project and maintaining a greater RBAE in the dorsal cortex to the end of the experiment. A significant difference between treatment groups occurred on day 0 (P=.05) and day 50 (P=.03).

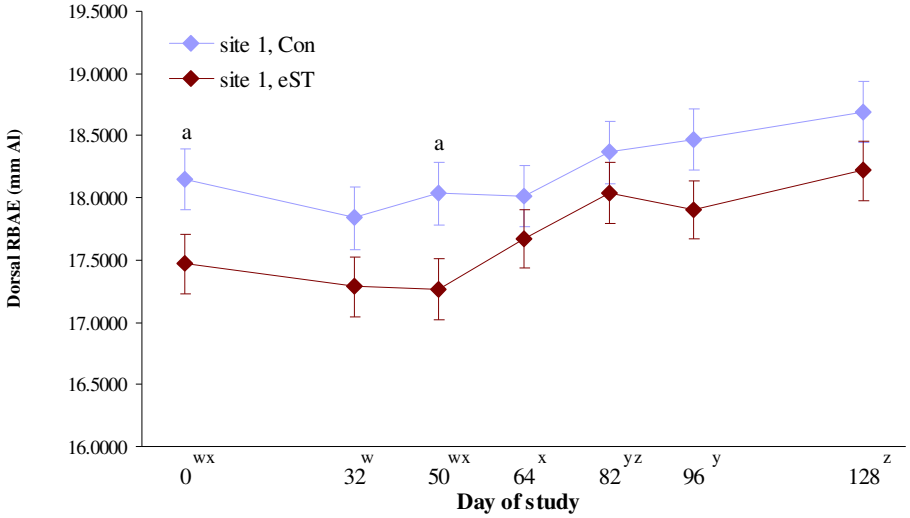


Figure 10. Dorsal radiographic bone aluminum equivalence (RBAE) (mm Al) at site 1.
[a] Treatments differ (P≤.05).
[wxyz] Days not sharing the same superscript differ (P≤.05).

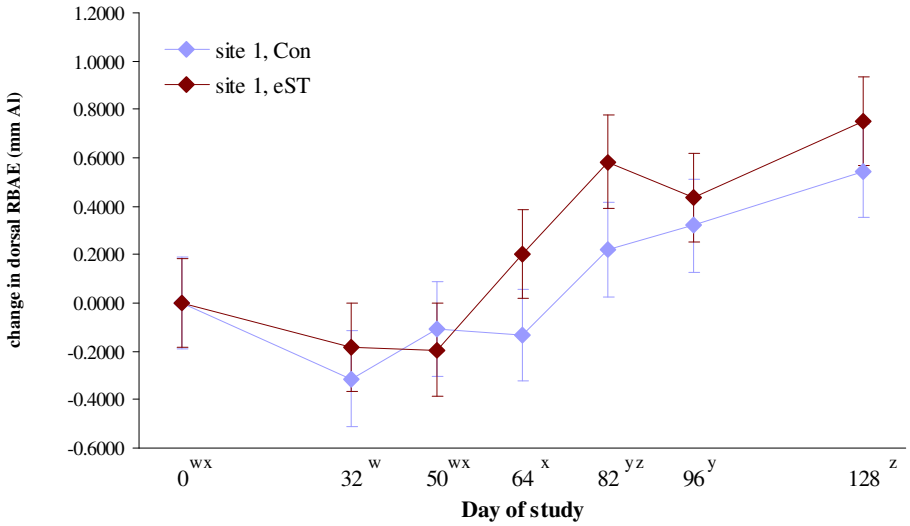


Figure 11. Normalized dorsal radiographic bone aluminum equivalence (RBAE) (mm Al) at site 1.
[wxyz] Days not sharing the same superscript differ (P≤.05).

Dorsal RBAE data were normalized to day 0 (Figure 11, Tables A-4 and B-10). There was not a treatment difference or a treatment*day interaction. There was a significant day effect (P<.0001) with a pattern for the change in dorsal RBAE to decline from day 0 to day 32 (control group) or from day 0 to day 50 (eST group), and then an overall increase to the end of the trial, with temporary decreases on day 64 (control group), or day 96 (eST group).

Site 2

A significant day effect was seen in dorsal RBAE at site 2 (P<.0001), but there was not a treatment*day interaction or a significant treatment effect (Figure 12, Tables A-3 and B-11). The control horses had a trend for a greater amount of cortical bone in the dorsal cortex at site 2 than the eST horses (as measured by RBAE) at the start of the project on day 0 (P=.06), and maintained a greater RBAE in the dorsal cortex until day 96. By day 128, the eST horses had a greater RBAE in the dorsal cortex at site 2 than the control horses, but this difference was not statistically significant. A significant difference did occur between treatment groups on day 50 (P<.05).

Data were normalized to day 0 and reevaluated to observe any differences in dorsal cortical bone deposition at site 2 over the duration of the experiment (Figure 13, Tables A-4 and B-12). Day effects were significant (P<.0001), but no significant treatment or treatment*day effects occurred. Both treatment groups had an initial decrease in dorsal RBAE at site 2. The eST group recovered to above day 0 values in RBAE by day 64, but the control group did not rise above day 0 values until day 82. The eST group had a moderate decrease in dorsal RBAE from day 82 to day 96 that was not seen in the control group. Both groups had a similar increase in dorsal RBAE from day 96 to day 128. The change in dorsal RBAE was greater in the eST group than in the control group from day 64 to the end of the study, though this difference was not statistically significant. The increase in dorsal RBAE from day 0 values was significant in the control group on day 128 (P<.05), while in the eST group the increase was significant by day 82 (P=.02) and remained significantly different from day 0 through the end of the trial.

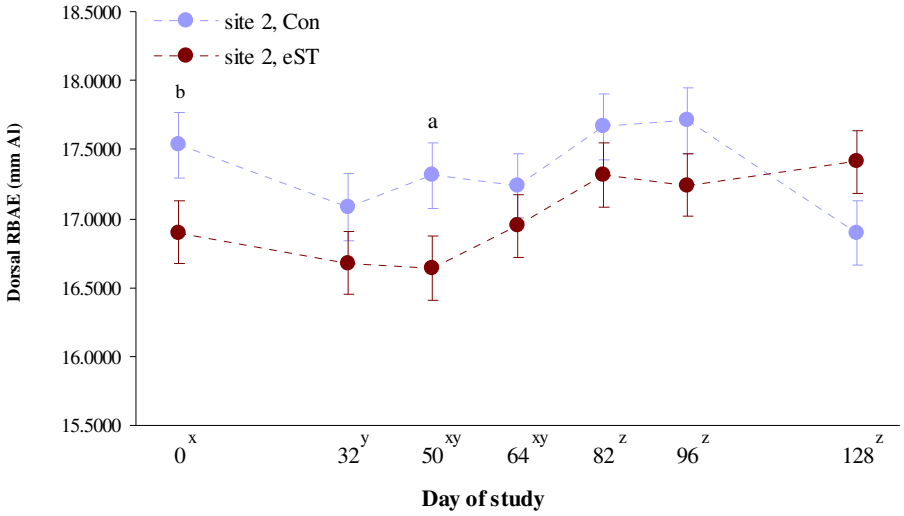


Figure 12. Dorsal radiographic bone aluminum equivalence (RBAE) (mm Al) at site 2.

[a] Treatments differ (P≤.05).

[b] Trend for treatments to differ (P≤.10).

[xyz] Days not sharing the same superscript differ (P≤.05).

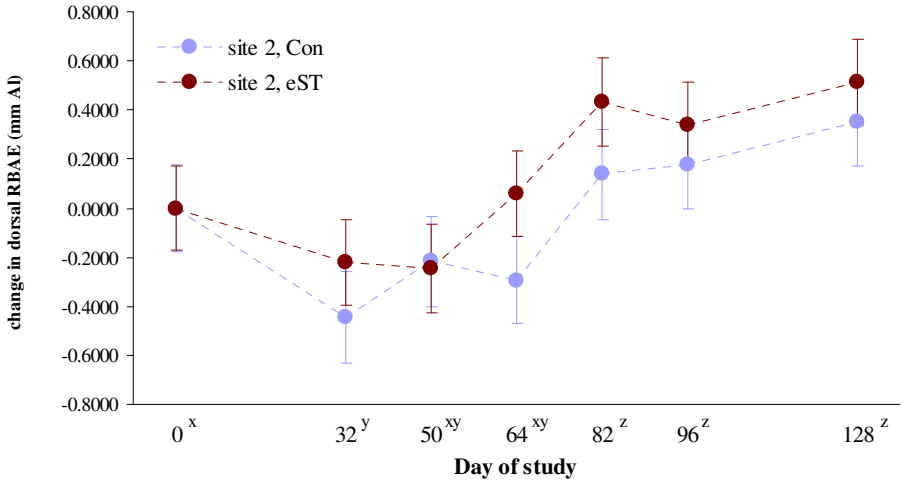


Figure 13. Normalized dorsal radiographic bone aluminum equivalence (RBAE) (mm Al) at site 2.

[xyz] Days not sharing the same superscript differ (P≤.05).

Site 3

There was a significant day effect (P<.0001) and a trend for treatment effect (P<.10) on dorsal RBAE at site 3, but there was not a day*treatment interaction (Figure 14, Tables A-3, B-13). Overall there was a pattern of significant decrease in dorsal RBAE from the start of the trial to day 32 (P<.01). There was a gradual gain in dorsal RBAE from day 32 to the end of the study. Values for dorsal RBAE were significantly greater than day 0 on day 82 (P<.05) and day 128 (P=.0004), and significantly greater (P<.0001) than day 32 on days 82, 96, and 128. Treatments were significantly different on day 0 (P=.05), and tended to be different on day 50 (P=.100). Both treatment groups had a decrease in dorsal RBAE from day 0 to day 32, then a gain in RBAE from day 32 to day 82. A minor decrease in dorsal RBAE from day 82 to day 96 was followed by a minor increase in dorsal RBAE from day 96 to day 128. The control group did show a minor decrease in RBAE from day 50 to day 64 that did not occur in the eST treatment group.

Data were normalized to day 0 and reevaluated to observe any differences in dorsal cortical bone deposition at site 3 over the duration of the experiment (Figure 15, Tables A-4 and B-14). Day effects were significant (P<.0001), but no significant treatment or treatment*day effects occurred. There was a significant decrease in dorsal RBAE from day 0 to day 32 (P=.01) and then an increase from day 32 to day 82. The increase in dorsal RBAE on day 82 was significant when compared to day 0 (P=.04) and day 32 (P<.0001). Another decrease in dorsal RBAE occurred from day 82 to day 96, followed by an increase in RBAE from day 96 to the end of the trial period.

The Control group of horses had a greater loss of bone mineral density as measured by RBAE from day 0 to day 32 than did the eST treatment group of horses. A gain in RBAE seen in the eST horses from day 50 to day 64 was not seen in the control horses. However, neither of these differences was large enough to be statistically significant.

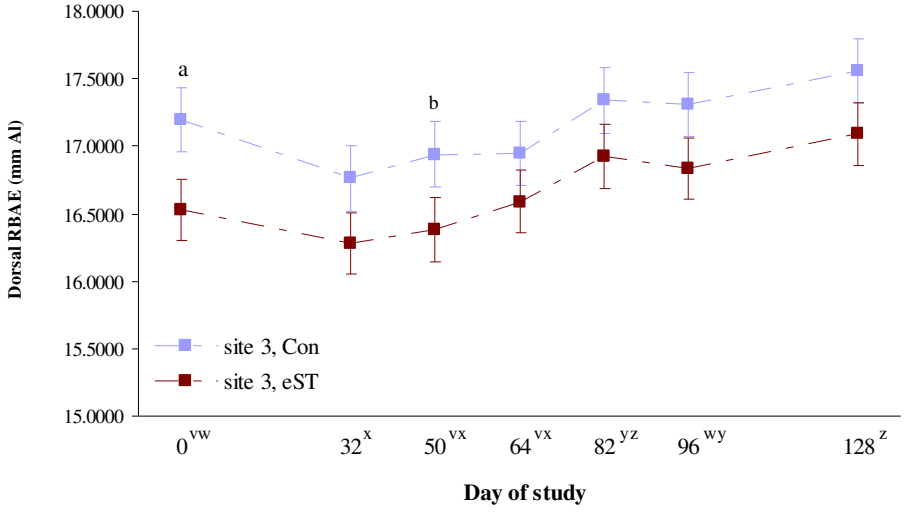


Figure 14. Dorsal radiographic bone aluminum equivalence (RBAE) (mm Al) at site 3.

[a] Treatments differ (P≤.05).

[b] Trend for treatments to differ (P≤.10).

[vwxyz] Days not sharing the same superscript differ (P≤.05).

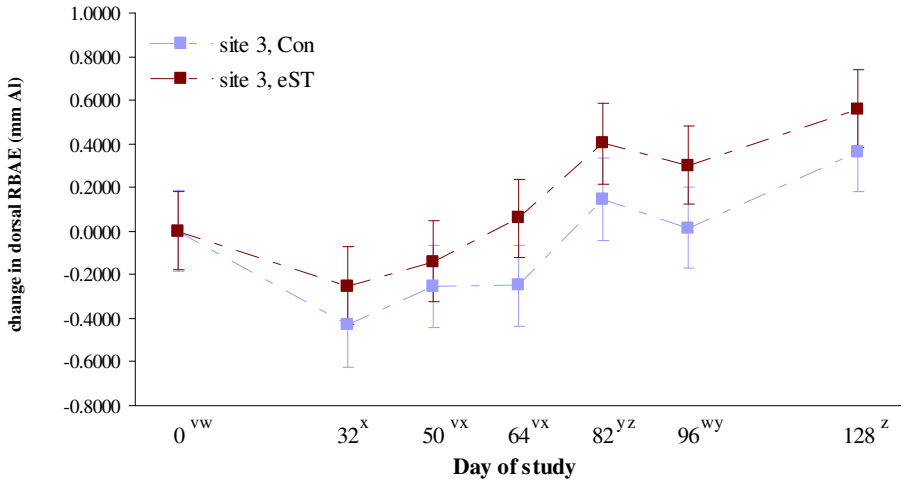


Figure 15. Normalized dorsal radiographic bone aluminum equivalence (RBAE) (mm Al) at site 3.

[vwxyz] Days not sharing the same superscript differ (P≤.05).

Site 4

There was a significant day effect (P<.0001), but no treatment effect or day*treatment interaction on dorsal RBAE at site 4 (Figure 16, Tables A-3, B-15). After a decrease in RBAE from day 0 to day 32, RBAE increased from day 32 to day 128.

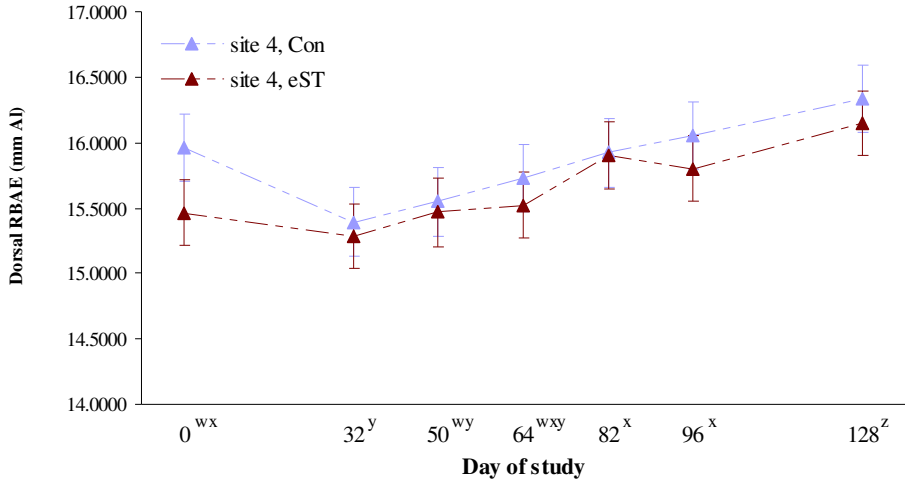


Figure 16. Dorsal radiographic bone aluminum equivalence (RBAE) (mm Al) at site 4. ^{wxyz} Days not sharing the same superscript differ (P≤.05).

The Control group of horses started the project with a non-significantly greater amount of dorsal RBAE at site 4 than the eST group. The difference seen between the two groups on day 0 had decreased by day 32.

Data were normalized to day 0 and reevaluated to observe any differences in dorsal cortical bone deposition at site 4 over the duration of the experiment (Figure 17, Tables A-4 and B-16). The significant decrease in dorsal RBAE that occurred from day 0 to day 32 (P=.02) was more pronounced in the Con group than it was in the eST group, but there were no significant differences observed between the two groups in dorsal RBAE at site 4. Though the two treatment groups appeared to differ on day 82 in Figure 17, they were not statistically different (P=.101). Both groups had an increase in RBAE

from day 32 until day 82. From day 82 to day 96 the eST group had a decrease in RBAE that was not seen in the Con group. Day 128 dorsal RBAE values were significantly greater than those seen on days 0 (P=.0009), 32 (P<.0001), 50 (P<.0001), and 64 (P=.0001).

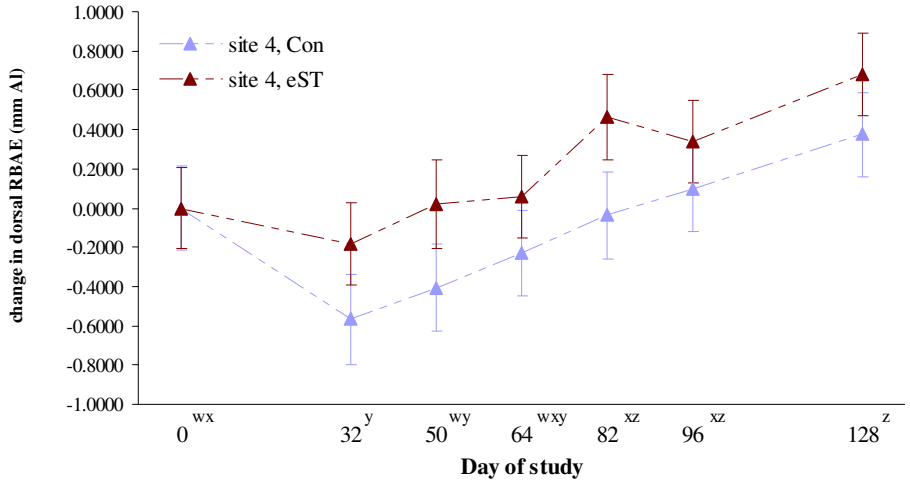


Figure 17. Normalized dorsal radiographic bone aluminum equivalence (RBAE) (mm Al) at site 4.

^{wxyz} Days not sharing the same superscript differ (P≤.05).

Lateral RBAE

Site 1

The lateral RBAE at site 1 was significantly affected by day (P<.0001) but not by treatment or by day*treatment interaction (Figure 18, Tables A-5 and B-17). Except for a decline in measured RBAE from day 32 to day 50, lateral RBAE at site 1 showed a slow but steady increase throughout the experiment. This increase resulted in significant differences between day 0 and day 96 (P=.008) and between day 0 and day 128 (P<.0001) in lateral RBAE.

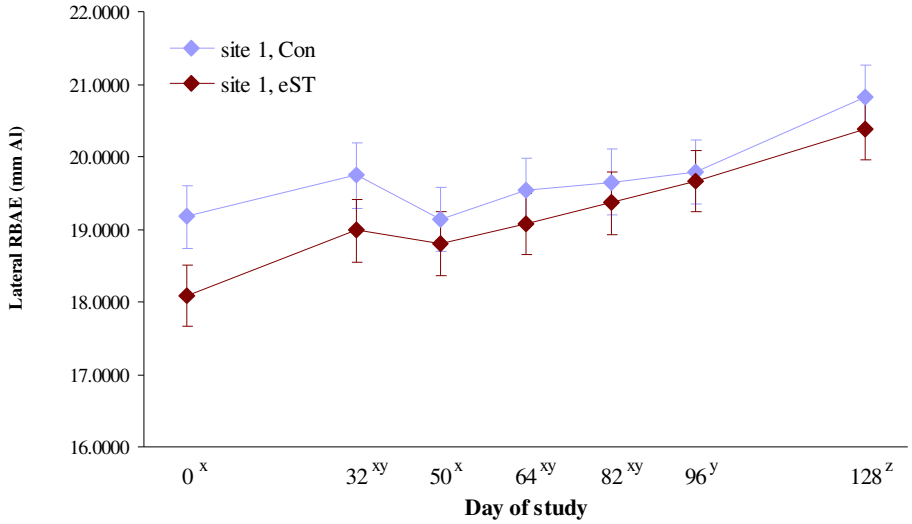


Figure 18. Lateral radiographic bone aluminum equivalence (RBAE) (mm Al) at site 1. ^{xyz} Days not sharing the same superscript differ (P≤.05).

Data were normalized to day 0 to better observe and evaluate any differences in lateral cortical bone deposition at site 1 over the duration of the experiment (Figure 19, Tables A-6 and B-18). This increase in lateral RBAE at site 1 resulted in significant differences between day 0 and both day 96 (P=.008) and day 128 (P<.0001), and between day 96 and day 128 (P=.002). Day effects were significant (P<.0001), but no differences due to treatment or day*treatment interactions occurred. On day 0 the Con group of horses had a non-significantly greater RBAE in the lateral cortex than the eST group. This difference between the two groups decreased over the course of the trial, but did not entirely disappear.

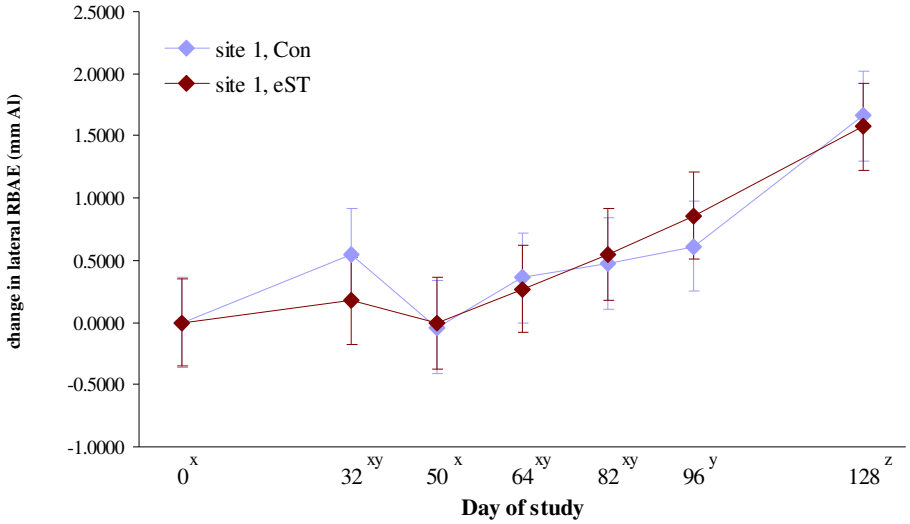


Figure 19. Normalized lateral radiographic bone aluminum equivalence (RBAE) (mm Al) at site 1.

^{xyz} Days not sharing the same superscript differ (P≤.05).

Site 2

There was a significant day effect (P<.0001), but no treatment effect or day* treatment interaction on RBAE in the lateral cortex at site 2 (Figure 20, Tables A-5 and B-19). There was an increase in RBAE from day 0 to day 32, then a decrease in RBAE from day 32 to day 50. A gradual but steady increase in RBAE continued from day 50 to the end of the experiment on day 128. Days 82, 96, and 128 all had a significantly greater RBAE in the lateral cortex than did day 0 (P=.04, P=.07, and P<.0001 respectively). The increase in RBAE from day 96 to day 128 was significant (P=.0008).

Data were normalized to day 0 values and re-evaluated (Figure 21, Tables A-6 and B-20). Once again day effects were significant (P<.0001), but treatment effects and day*treatment interactions did not effect lateral RBAE at site 2.

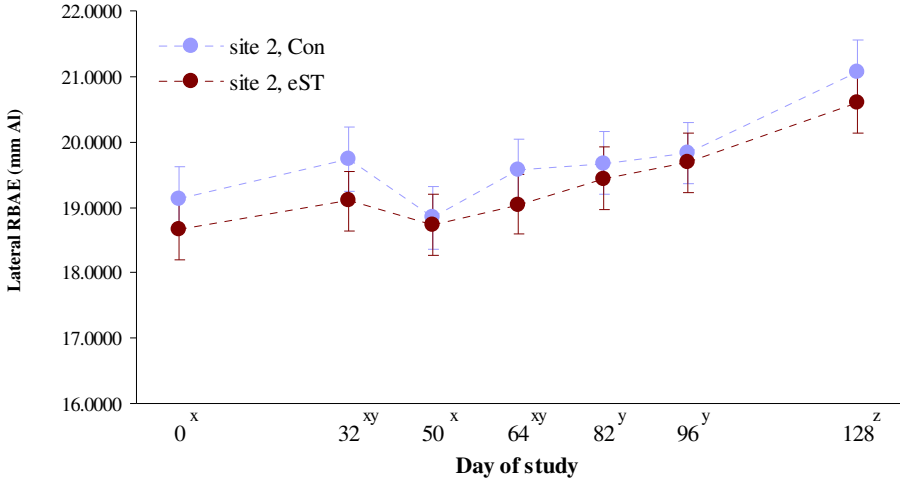


Figure 20. Lateral radiographic bone aluminum equivalence (RBAE) (mm Al) at site 2.
^{xyz} Days not sharing the same superscript differ (P≤.05).

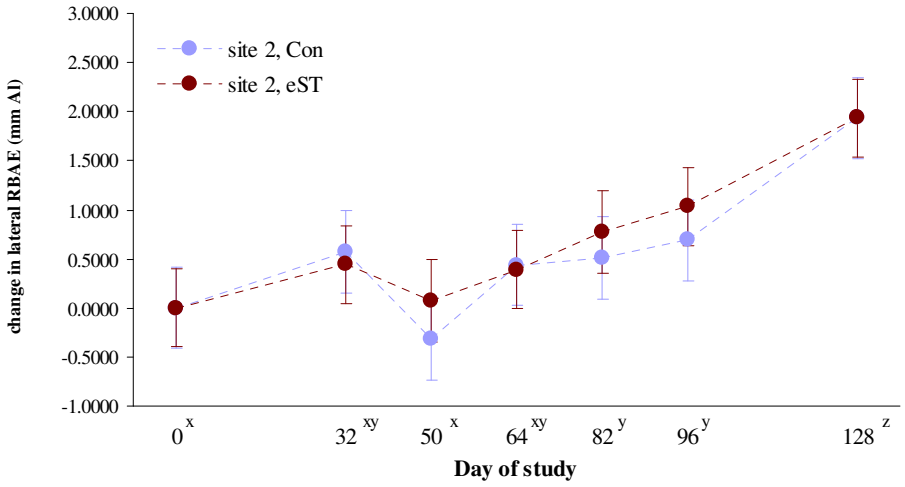


Figure 21. Normalized lateral radiographic bone aluminum equivalence (RBAE) (mm Al) at site 2.
^{xyz} Days not sharing the same superscript differ (P≤.05).

Site 3

Lateral RBAE at site 3 was significantly effected by day (P<.0001), but not by treatment or by day*treatment interaction (Figure 22, Tables A-5 and B-21). The pattern of change in lateral RBAE at site 3 was very similar to that seen at site 2. There was an increase in RBAE from day 0 to day 32, a decrease in RBAE from day 32 to day 50, and finally an increase in RBAE from day 50 to day 128. Day 96 lateral RBAE was significantly greater than day 0 (P=.01). Day 128 lateral RBAE at site 3 was significantly greater than day 96 (P=.0004) and day 0 (P<.0001). Treatment groups were not different on any day.

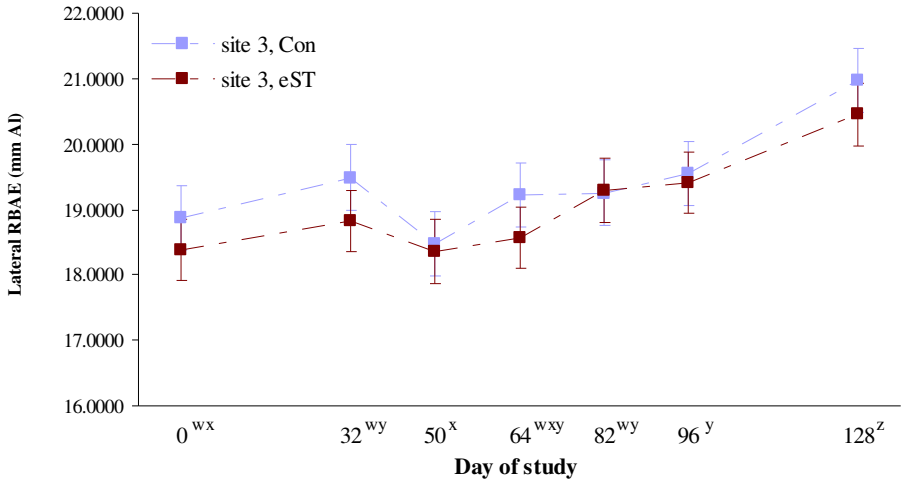


Figure 22. Lateral radiographic bone aluminum equivalence (RBAE) (mm Al) at site 3. ^{wxyz} Days not sharing the same superscript differ (P≤.05).

Data were normalized to better evaluate changes that occurred over time in lateral RBAE at site 3. Day effects were significant (P<.0001), but no treatment effects or day*treatment interactions were observed (Figure 23, Tables A-6 and B-22). Lateral RBAE increased from day 0 to day 32, decreased significantly from day 32 to day 50 (P=.03), and increased from day 50 to day 128. Day 96 values were significantly greater

than those on day 0 (P=.01). Day 128 values were significantly greater than those on day 0 (P<.0001) or day 96 (P=.0004). The decrease in lateral RBAE at site 3 that occurred from day 32 to day 50 was more pronounced in the Con group of horses, though the difference between the two groups was not significant. By day 128 there was no difference in lateral RBAE at site 3 between the two treatment groups.

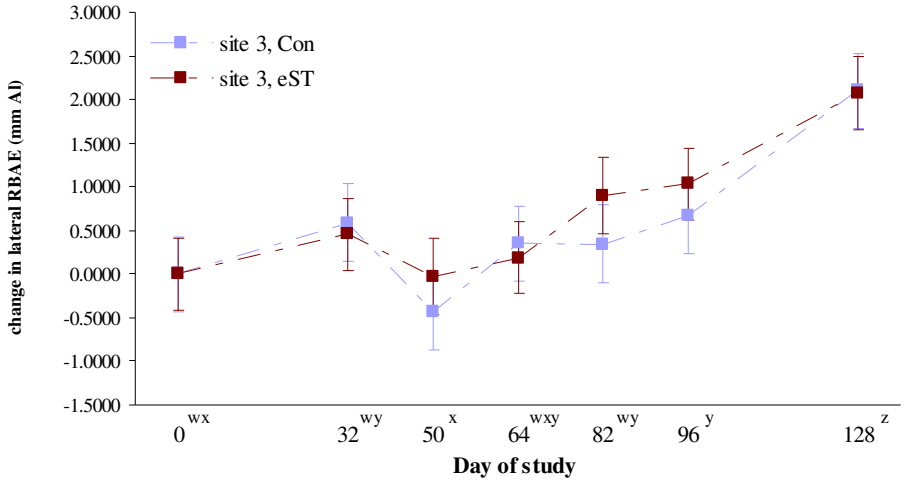


Figure 23. Normalized lateral radiographic bone aluminum equivalence (RBAE) (mm Al) at site 3.

[wxyz] Days not sharing the same superscript differ (P≤.05).

Site 4

Lateral RBAE at site 4 was significantly affected by day (P<.0001) but not significantly affected by treatment or by day*treatment interaction (Figure 24, Tables A-5 and B-23). Lateral RBAE increased from day 0 to day 32, decreased from day 32 to day 50, and increased from day 50 through the end of the trial. By day 82, lateral RBAE at site 4 was significantly greater than the initial measurements taken on day 0 (P=.04). Day 128 RBAE values of 19.02 mm aluminum were significantly greater than those seen on any of the other sampling dates.

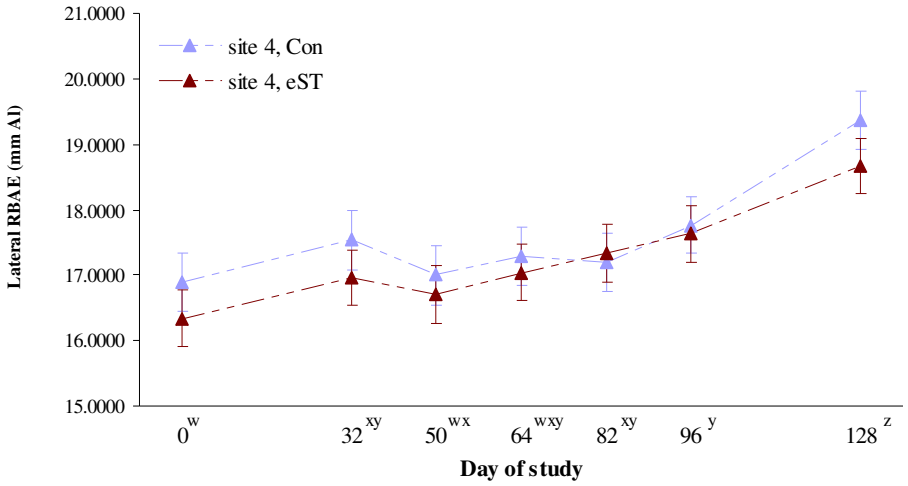


Figure 24. Lateral radiographic bone aluminum equivalence (RBAE) (mm Al) at site 4.
^{wxyz} Days not sharing the same superscript differ (P≤.05).

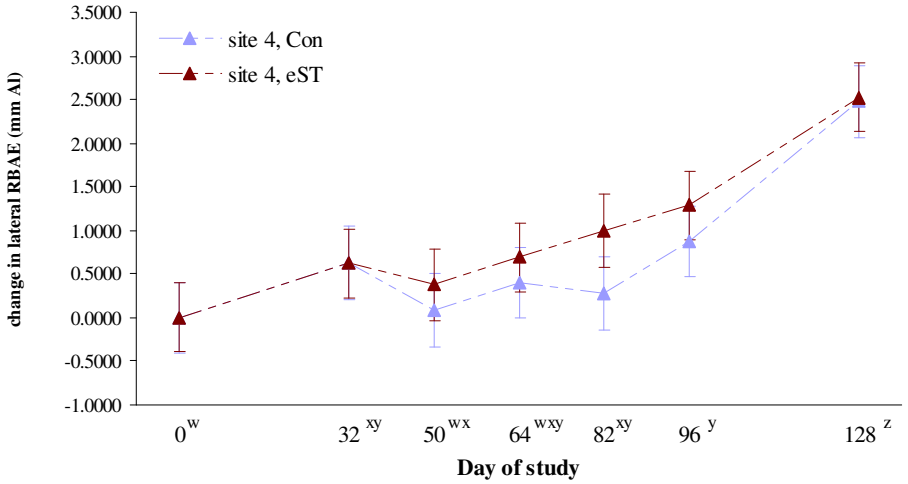


Figure 25. Normalized lateral radiographic bone aluminum equivalence (RBAE) (mm Al) at site 4.
^{wxyz} Days not sharing the same superscript differ (P≤.05).

Data were normalized to better view changes in lateral RBAE at site 4 that occurred over the course of this experiment (Figure 25, Tables A-6 and B-24). There was a day effect (P<.0001) but not a significant treatment effect or day*treatment interaction.

Medial RBAE

Site 1

There was a significant day effect (P<.0001) but no treatment effect or day*treatment interaction in RBAE in the medial cortex at site 1 (Figure 26, Tables A-7 and B-25). There was a pattern of steady increase in RBAE from day 0 to the end of the trial on day 128, with the exception of a minor decrease in RBAE on day 96. Day 32 values were significantly greater than those seen on day 0 (P=.006). Increases seen from day 32 to day 64 were not significant until day 82 values, which were significantly greater than those seen on day 32 (P<.0001), day 50 (P=.0004), and day 64 (P=.02). A decrease in medial RBAE at site 1 from day 82 to day 96 was followed by a significant increase in RBAE by day 128. Day 128 RBAE was significantly greater than day 82 (P=.01) and day 96 (P=.002). A non-significant difference between the two treatment groups on day 0 had totally disappeared by day 96. No significant differences were seen between the two treatment groups at this site.

Medial RBAE data from site 1 were normalized to better evaluate changes in RBAE over the course of the trial (Figure 27, Tables A-8 and B-26). Day effects were significant (P<.0001). No treatment effects or day*treatment interactions were seen. The increase in RBAE from day 0 to day 32 was significant (P=.006). From day 32 to day 50 medial RBAE continued to increase in the eST treatment group, but decreased in the Con group. Both groups had a gain in RBAE from day 50 to day 64 and on to day 82. Day 82 was significantly greater than day 64 (P=.02). Both groups had a decrease in RBAE from day 82 to day 96 and then an increase in RBAE from day 96 to day 128. Day 128 medial RBAE at site 1 was significantly greater than day 96 (P=.002), day 82 (P=.01), and days 0, 32, 50, and 64 (all P<.0001). The eST group of horses had a non-significant greater increase in RBAE than the Con group of horses.

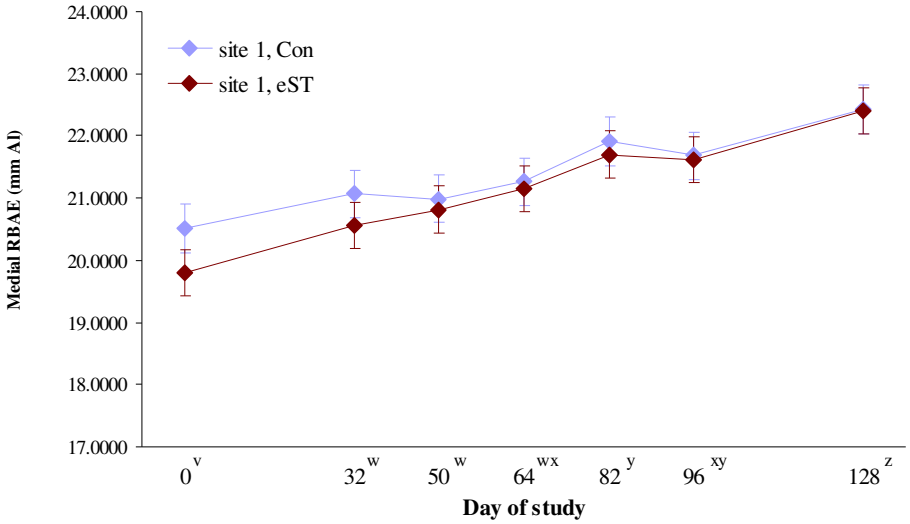


Figure 26. Medial radiographic bone aluminum equivalence (RBAE) (mm Al) at site 1.
^{vwxyz} Days not sharing the same superscript differ (P≤.05).

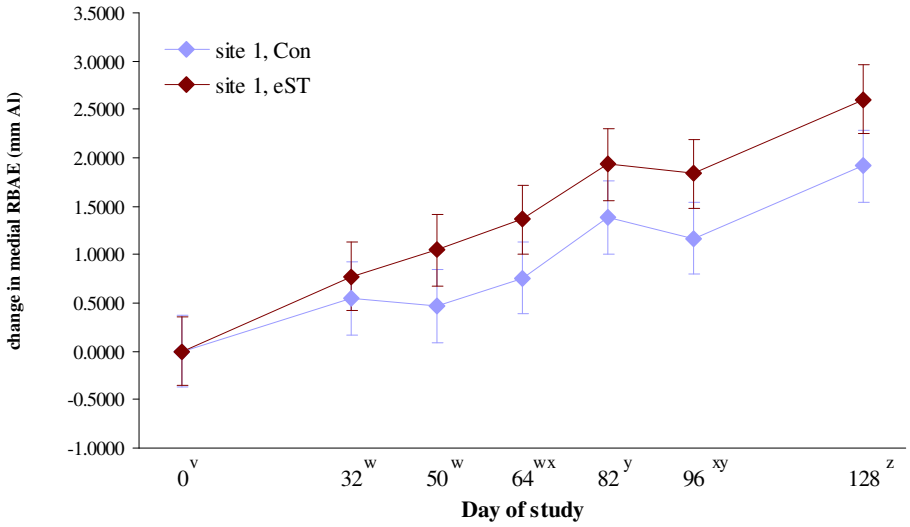


Figure 27. Normalized medial radiographic bone aluminum equivalence (RBAE) (mm Al) at site 1.
^{vwxyz} Days not sharing the same superscript differ (P≤.05).

Site 2

At site 2, medial RBAE day effects were significant (P<.0001) but no treatment effects or day*treatment interactions occurred (Figure 28, Tables A-7 and B-27). The increase in RBAE from day 0 to day 32 was significant (P=.003). Further increases were not statistically significant until day 82, which had a greater RBAE value than did day 32 (P=.0002), day 50 (P=.0007), or day 64 (P=.02). A small decrease in RBAE was seen from day 82 to day 96, followed by a significant increase in RBAE from day 96 to day 128 (P=.001).

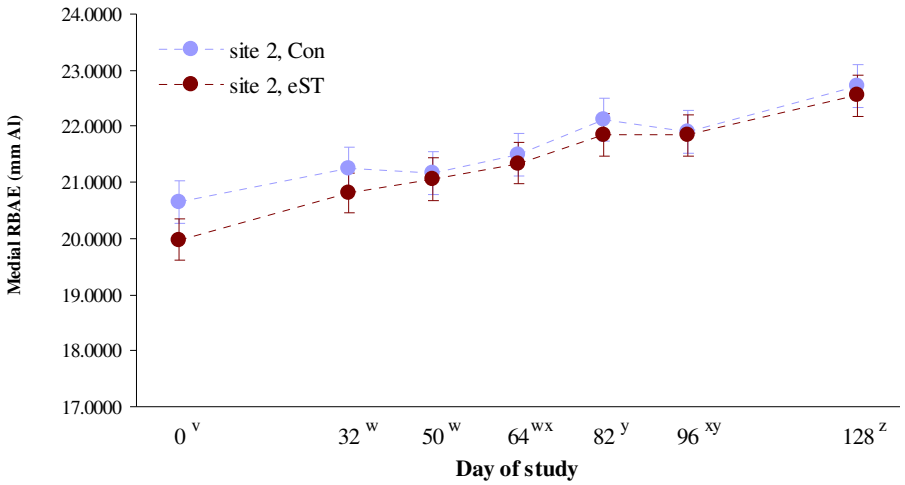


Figure 28. Medial radiographic bone aluminum equivalence (RBAE) (mm Al) at site 2. ^{vwxyz} Days not sharing the same superscript differ (P≤.05).

Data were normalized to day 0 values (Figure 29, Tables A-8 and B-28). Change in medial RBAE at site 2 was significantly affected by day (P<.0001), but not by treatment or day*treatment interaction. Measured RBAE on day 32 was significantly greater than on day 0 (P=.004). The gain in RBAE continued in the eST group to day 82. There was a non-significant loss in RBAE from day 32 to day 50, then a gain in RBAE from day 50 to day 82 in the Con group. A loss in RBAE occurred in both treatment groups from day

82 to day 96, followed by a gain in RBAE from day 96 to day 128. The medial RBAE measurements at site 2 on day 128 were significantly greater than that seen on day 0 (P<.0001), day 32 (P<.0001), day 50 (P<.0001), day 64 (P<.0001), day 82 (P=.009), and day 96 (P=.001). Differences seen between the two treatment groups were not significant.

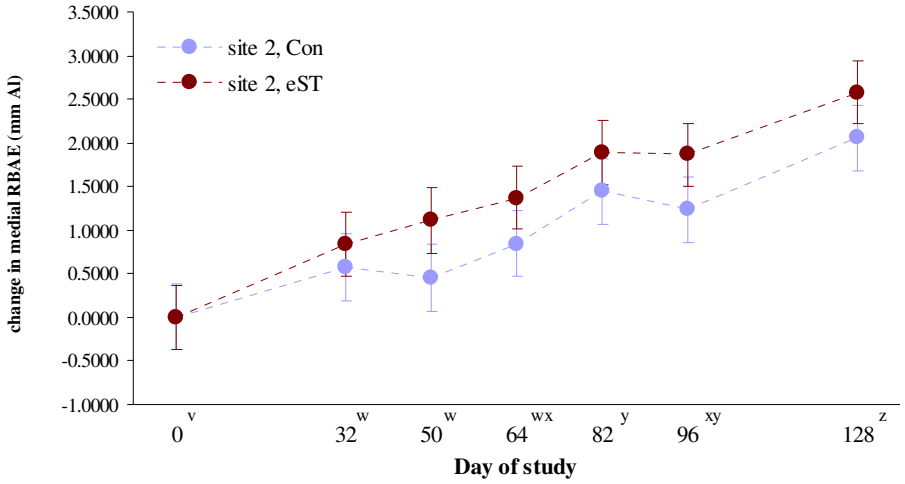


Figure 29. Normalized medial radiographic bone aluminum equivalence (RBAE) (mm Al) at site 2.

[vwxyz] Days not sharing the same superscript differ (P≤.05).

Site 3

Medial RBAE at site 3 was significantly affected by day (P<.0001), but not by treatment or day*treatment interaction (Figure 30, Tables A-7 and B-29). Overall, an increase in RBAE occurred from day 0 to day 82 and from day 96 to day 128. From day 82 to day 96 a decrease in RBAE was seen. Within the treatment groups, the non-significantly greater RBAE in the Con group compared to the eST group on day 0 had disappeared by day 64.

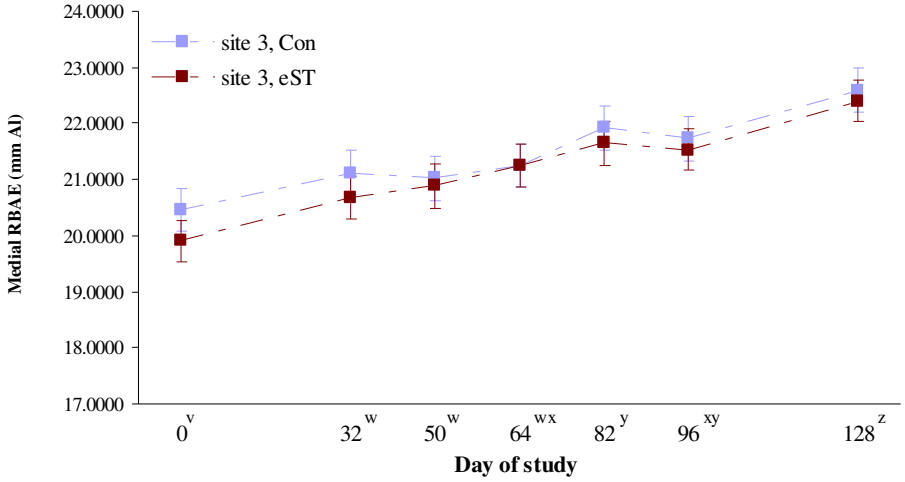


Figure 30. Medial radiographic bone aluminum equivalence (RBAE) (mm Al) at site 3.
^{vwxyz} Days not sharing the same superscript differ (P≤.05).

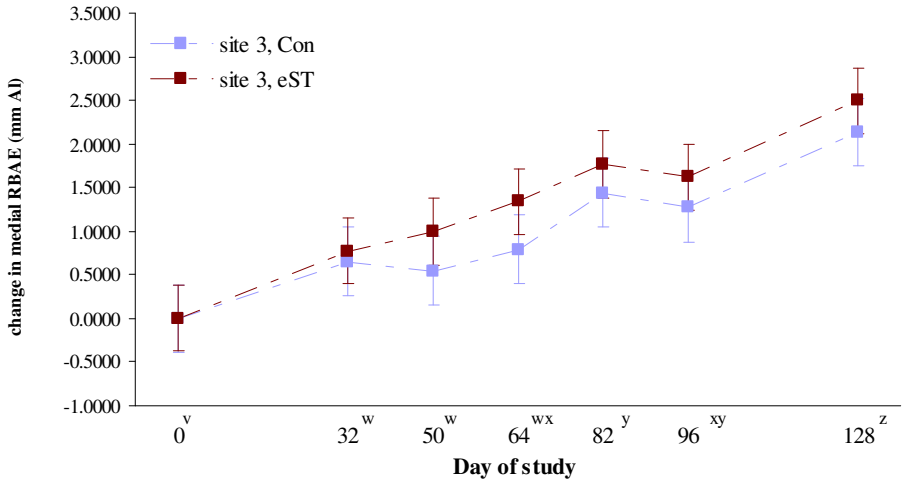


Figure 31. Normalized medial radiographic bone aluminum equivalence (RBAE) (mm Al) at site 3.
^{vwxyz} Days not sharing the same superscript differ (P≤.05).

Data were normalized to day 0 and re-evaluated. Once again, changes in medial RBAE at site 3 were significantly affected by day (P<.0001) but not by treatment or

day*treatment interaction (Figure 31, Tables A-8, B-30). More apparent in the normalized data than in the raw data was the non-significant, but persistently greater RBAE in the eST group than in the Con group. This difference was first apparent on day 50 and continued through the end of the trial.

Site 4

Medial RBAE at site 4 was significantly affected by day (P<.0001) but not by treatment or day*treatment interaction (Figure 32, Tables A-7 and B-31). Day 0 RBAE was significantly less than day 32 RBAE (P=.003). A non-significant difference in RBAE between the two treatment groups on day 0 had been eliminated by day 64 due to the eST group experiencing a greater increase in RBAE than the Con group. The eST group continued to show a more rapid increase in medial RBAE at site 4 than the Con group from day 64 to day 82. However, from day 82 to day 96 the eST group had a decrease in RBAE at the same time that the Con group had a gain in RBAE. After day 96 both groups increased in RBAE to the end of the trial on day 128. As a result, the eST group had a higher RBAE than the Con group only on day 82.

Normalized data were re-evaluated to better observe changes over time in medial RBAE at site 4 (Figure 33, Tables A-8 and B-32). Day effects were significant (P<.0001), but no treatment or day*treatment interactions were seen. The greater gain in RBAE in the eST group compared to the Con group was most pronounced on day 82, but was not significant (P=.101). In spite of a decrease in RBAE in the Con group from day 50 to day 64, and in the eST group from day 82 to day 96, there was a general gain in medial RBAE at site 4 over the course of the trial. Gain in RBAE caused day 50 to be significantly different than day 0 (P=.03), and day 128 to be significantly different than day 96 (P=.0004), day 82 (P=.06), day 64 (P=.0001), day 50 (P<.0001), day 32 (P<.0001), and day 0 (P<.0001).

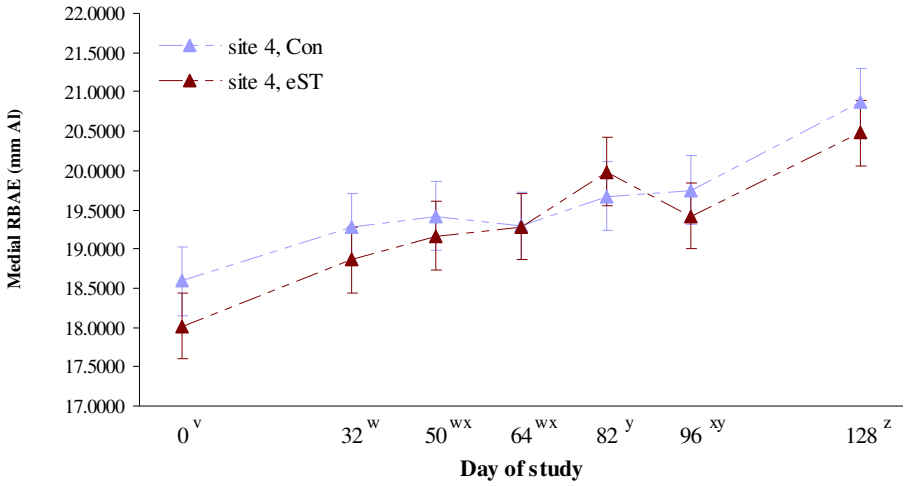


Figure 32. Medial radiographic bone aluminum equivalence (RBAE) (mm Al) at site 4.
^{vwxyz} Days not sharing the same superscript differ (P≤.05).

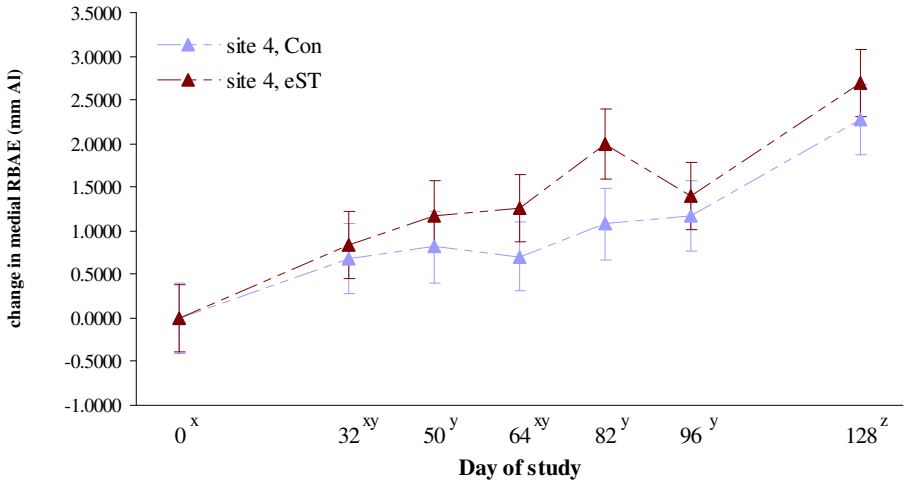


Figure 33. Normalized medial radiographic bone aluminum equivalence (RBAE) (mm Al) at site 4.
^{xyz} Days not sharing the same superscript differ (P≤.05).

Palmar RBAE

Site 1

There was an overall day effect on palmar RBAE at site 1 (P<.002) but there was no significant difference between treatment groups on any given day (Figure 34, Tables A-9 and B-33). Changes in palmar RBAE at site 1 were not affected significantly by treatment or day*treatment interaction. Palmar RBAE decreased significantly from day 0 to day 32 (P=.004). From day 32 to day 96 the Con group had a gradual and steady increase in RBAE that leveled off from day 96 to day 128. From day 32 to day 50 the eST group had decrease in RBAE, followed by an increase in RBAE from day 32 to day 82, another decrease in RBAE from day 82 to day 96, and ending the trial with an increase in RBAE from day 96 to day 128. The RBAE values on day 128 were not statistically different from day 0.

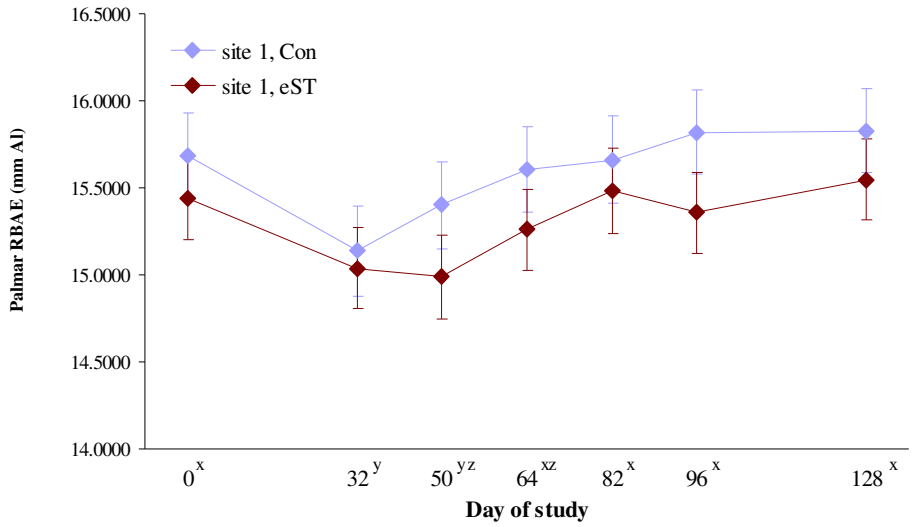


Figure 34. Palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 1. ^{xyz} Days not sharing the same superscript differ (P≤.05).

Palmar RBAE at site 1 data were normalized to day 0 and statistics were re-run to better observe changes in RBAE over time (Figure 35, Tables A-10 and B-34). Day effects were significant (P=.002). Neither treatment effects nor day*treatment interactions were significant. A significant loss of palmar RBAE at site 1 from day 0 to day 32 (P=.004), was followed by a gain in palmar RBAE from day 32 to day 128. Day 128 palmar RBAE data were not statistically different from day 0.

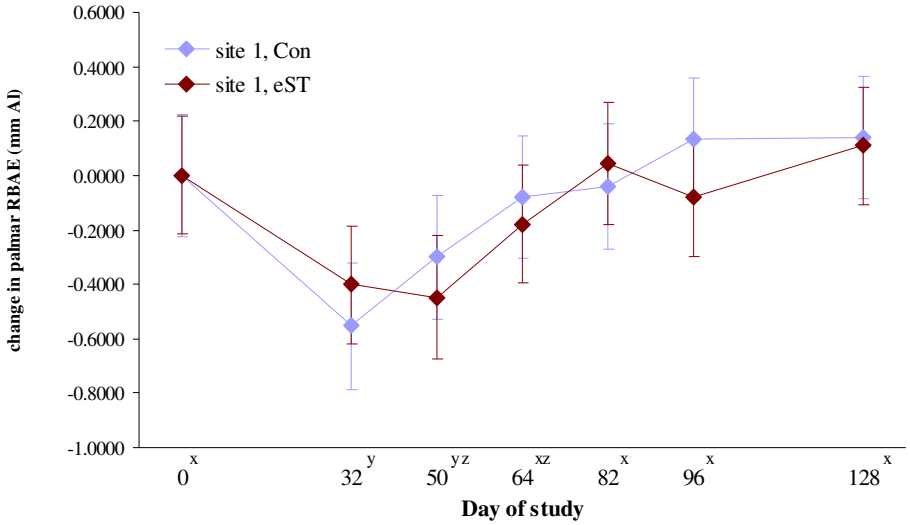


Figure 35. Normalized palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 1.

^{xyz} Days not sharing the same superscript differ (P≤.05).

Site 2

Palmar RBAE at site 2 was significantly affected by day (P=.004) but not by treatment or by day*treatment interaction (Figure 36, Tables A-9 and B-35). Bone mineral density, as measured by RBAE, decreased significantly from day 0 to day 32 (P=.006), and continued to decrease to day 50. From day 50 to day 128 palmar RBAE increased and by day 82 this increase resulted in RBAE values significantly greater than on day 32 (P=.04) and day 50 (P=.01), but not statistically different from day 0. The Con group of horses

had a non-significant greater palmar RBAE at site 2 on day 0 that continued to the end of the project.

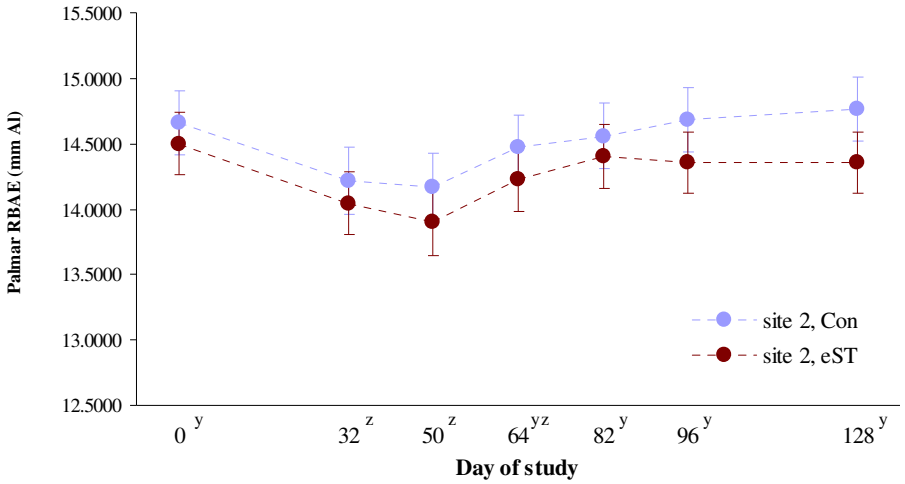


Figure 36. Palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 2. ^{yz} Days not sharing the same superscript differ (P≤.05).

Palmar RBAE data from site 2 were normalized and statistics re-run to more closely evaluate changes over time (Figure 37, Tables A-10 and B-36). Day effects were significant (P=.003) but no treatment effect or a day*treatment interaction occurred. There was a significant decrease in palmar RBAE from day 0 to day 32 (P=.006) that continued to day 50 (P=.001). From day 50 to day 128 palmar RBAE increased significantly (P=.001), but did not exceed day 0 values. The Con group of horses had a non-significantly less decrease in palmar RBAE than did the eST horses at day 50. The Con group also had a non-significantly greater increase in palmar RBAE from day 82 to day 128, which resulted in the Con group, but not the eST group exceeding day 0 values in palmar RBAE at site 2 on days 96 and 128.

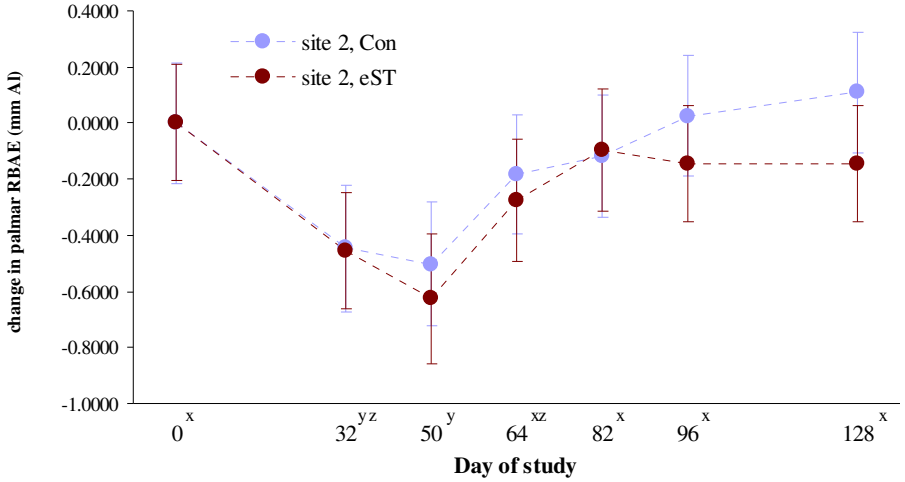


Figure 37. Normalized palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 2.

^{xyz} Days not sharing the same superscript differ (P≤.05).

Site 3

Palmar RBAE at site 3 was significantly affected by day (P=.01) but not by treatment or day*treatment interaction (Figure 38, Tables A-9 and B-37). Palmar RBAE at site 3 decreased significantly from day 0 to day 32 (P=.007) and continued to decrease to day 50, which was also significantly different from day 0 (P=.003). After day 50, palmar RBAE increased through the end of the project on day 128, but did not exceed day 0 values. A non-significantly larger palmar RBAE in the Con group than in the eST group continued through the project and increased from day 82 to day 96.

Data from palmar RBAE at site 3 were normalized and re-evaluated (Figure 39, Tables A-10 and B-38). Once again, day effects were significant (P=.01) and no treatment effects or day*treatment interaction were observed. A significant loss of bone mineral density as measured by RBAE occurred between day 0 and day 32 (P=.01) and between day 0 and day 50 (P=.002). A gain in palmar RBAE followed from day 50 to the end of the trial on day 128, with day 128 values not exceeding day 0 values. The Con group had a non-statistically greater gain in palmar RBAE at site 3 than the eST group from day 82 to day 96.

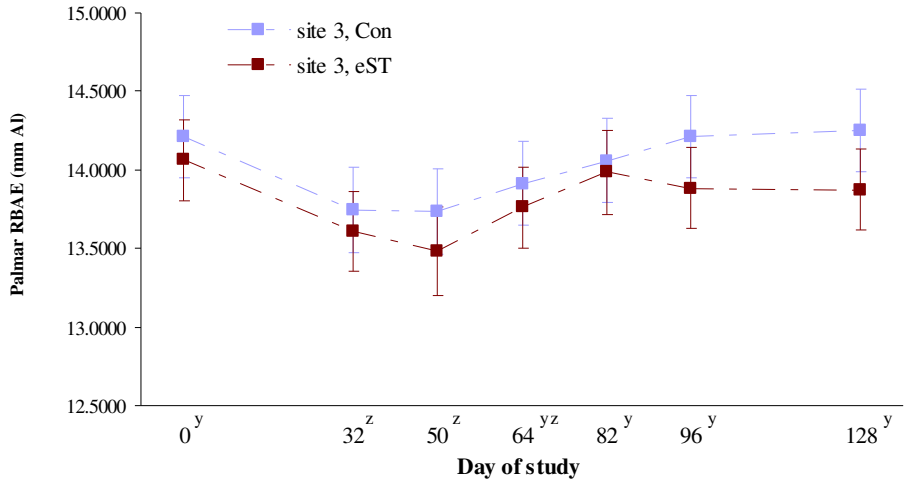


Figure 38. Palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 3.
^{yz} Days not sharing the same superscript differ (P≤.05).

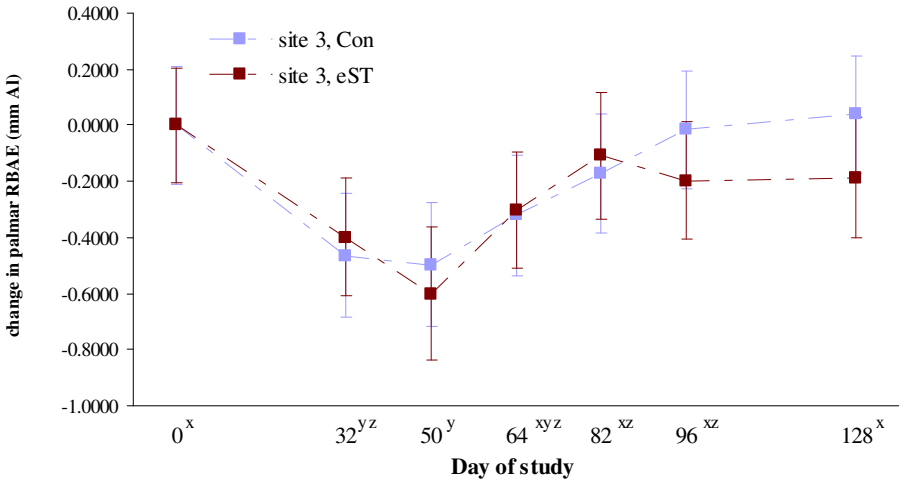


Figure 39. Normalized palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 3.
^{xyz} Days not sharing the same superscript differ (P≤.05).

Site 4

Palmar RBAE at site 4 had a trend for day effects (P=.07), but no effects occurred due to treatment or day*treatment interaction (Figure 40, Tables A-9 and B-39). A significant decrease in palmar RBAE took place between day 0 and day 32 (P=.02). Palmar RBAE increased from day 32 through day 128, and had increased enough to exceed day 0 values by day 96. The non-significantly larger bone density in the palmar cortex at site 4 seen in the Con group as compared to the eST group continued throughout the testing period, and increased in magnitude between day 82 and day 96 but was still not significant (P=.104).

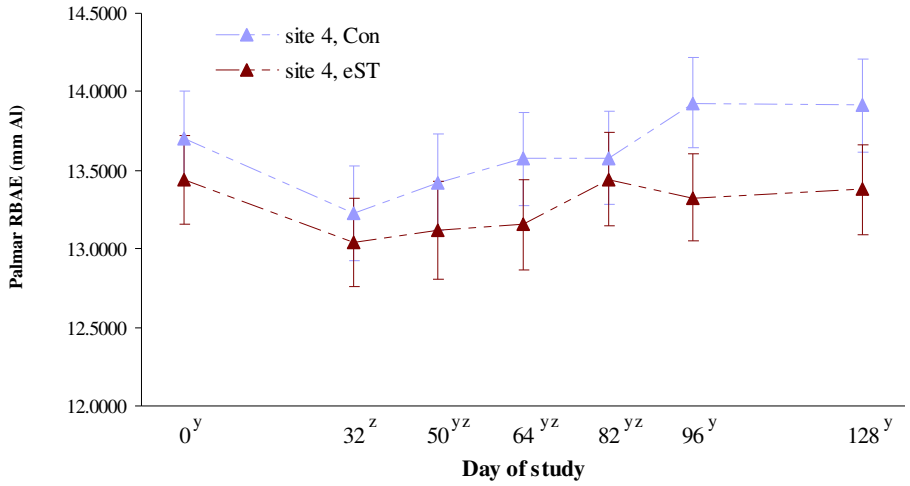


Figure 40. Palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 4.
^{yz} Days not sharing the same superscript differ (P≤.05).

Data from palmar RBAE at site 4 were normalized to day 0 (Figure 41, Tables A-10 and B-40). There was a trend for day effects (P=.09), but not for treatment effects or for day*treatment interactions. A decrease in RBAE in the palmar cortex from day 0 to day 32 was significant (P=.03). An increase in RBAE occurred from day 32 to day 128, with day 96 palmar cortical bone mineral density exceeding day 0 values, though there was no

statistical difference between day 0 and any of the measured days with the exception of the aforementioned day 32. Differences between the two treatment groups were not significant, nor were there a consistent pattern observed between the two groups in the palmar cortex at site 4.

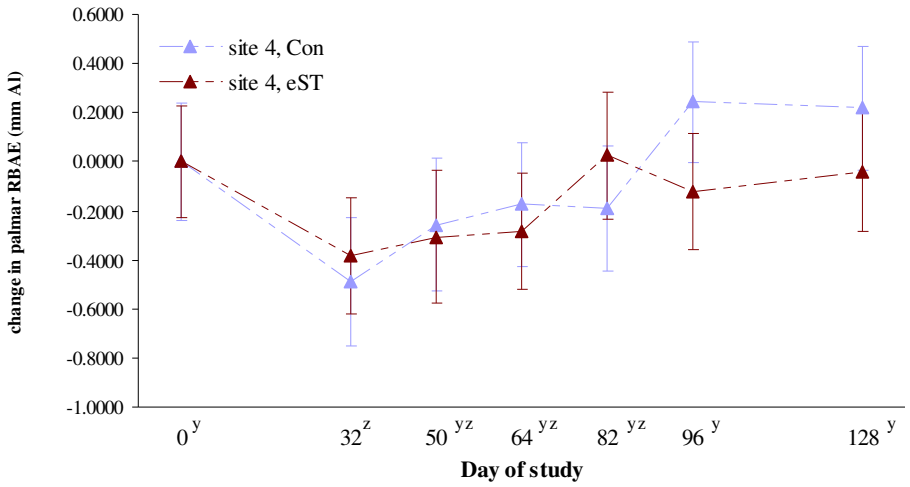


Figure 41. Normalized palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 4.

[yz] Days not sharing the same superscript differ (P≤.05).

Ratios of RBAE Measurements

Ratios of bone mineral density, as measured by RBAE, between various cortices were evaluated as this is an indication of bone remodeling. These ratios have been examined by Nielsen et al. (1997). Ratios examined and reported herein are dorsal/palmar, normalized dorsal/palmar, lateral/palmar, normalized lateral/palmar, medial/lateral, normalized medial/lateral, medial/palmar, and normalized medial/palmar.

Ratio of Dorsal to Palmar RBAE

Site 1

The ratio of dorsal to palmar RBAE at site 1 was significantly affected by day (P=.009) but not by treatment or by day*treatment interaction (Figure 42, Tables A-11 and B-41). From day 0 to day 32 the ratio of dorsal to palmar RBAE increased significantly (P=.02), then decreased from day 32 to day 64, but did not decrease to day 0 levels. There was a slight increase in the ratio of dorsal to palmar RBAE at site 1 measured on day 82, followed by a slight decrease on day 96, and then an additional slight increase on day 128. A non-significant difference between the two treatment groups in the ratio on day 0 had increased to be a trend by day 32 (P=.10). The difference between the two treatment groups then decreased until it was gone by day 64, as the eST group continued a pattern of a gradual increase in the ratio of dorsal to palmar RBAE through the entire trial while the Con group experienced an increase in the ratio, followed by a decrease in the ratio, before leveling off into a more consistent slow gain.

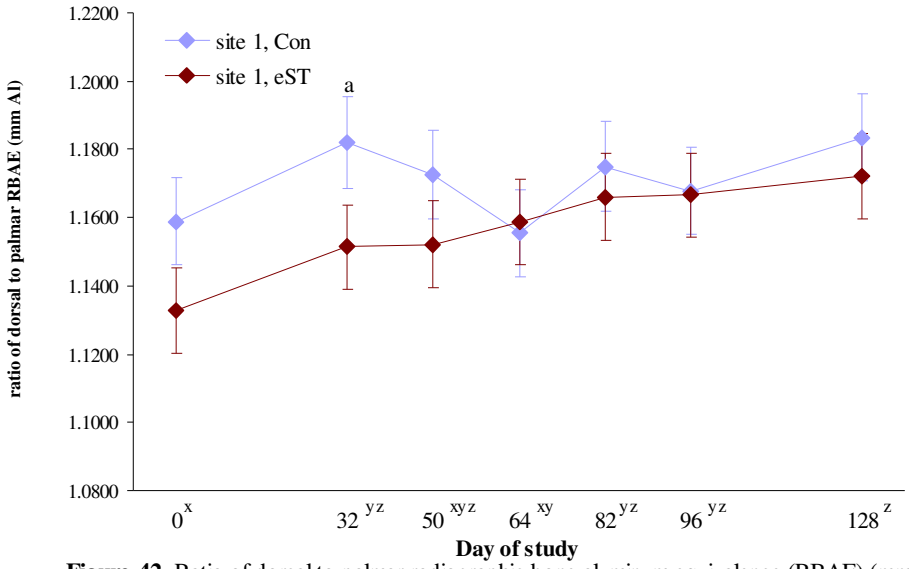


Figure 42. Ratio of dorsal to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 1.

[a] Trend for treatments to differ (P≤.10).

[xyz] Days not sharing the same superscript differ (P≤.05).

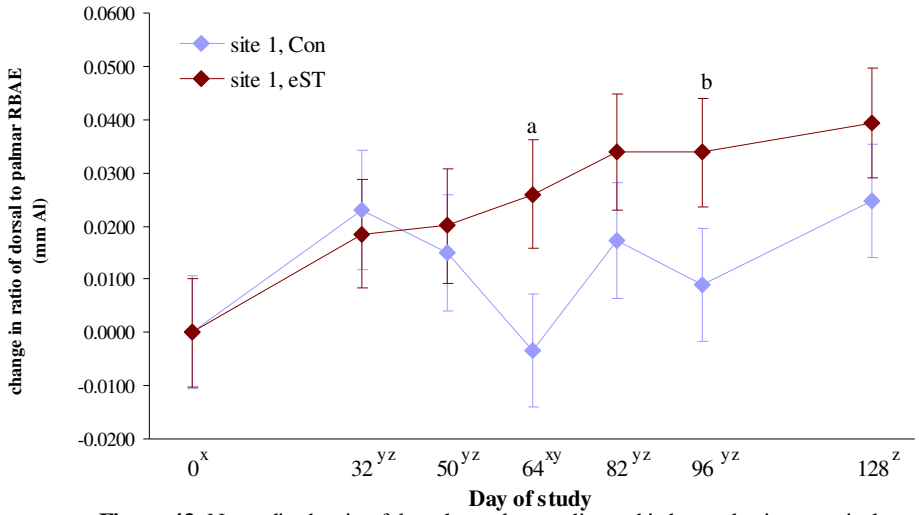


Figure 43. Normalized ratio of dorsal to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 1.

[a] Treatments differ (P≤.05).

[b] Trend for treatments to differ (P≤.10).

[xyz] Days not sharing the same superscript differ (P≤.05).

The data were normalized to day 0 and re-evaluated to better observe the change in the ratio of dorsal to palmar RBAE at site 1 over time (Figure 43, Tables A-12 and B-42). Day effects were significant (P=.008). There were no significant treatment effects or a day*treatment interaction. The ratio increased significantly from day 0 to day 32 (P=.02). From day 32 to day 64 the ratio decreased non-significantly. The increase in normalized dorsal/palmar RBAE from day 64 to day 128 was significant (P=.01). The eST group of horses displayed a fairly consistent increase in the ratio of dorsal to palmar RBAE from day 0 to day 128. In contrast, the Con group of horses had a gain in the ratio from day 0 to day 32, a significant loss in the ratio from day 32 to day 64 (P=.04), a gain in the ratio that trended toward significance from day 64 to day 82 (P=.09), a non-statistically important loss in the ratio from day 82 to day 96, and a non-significant gain in the ratio from day 96 to day 128. The two treatment groups were significantly

different from each other on day 64 (P=.05) and had a trend to be different from each other on day 96 (P=.100).

Site 2

At site 2 the ratio of dorsal to palmar RBAE was significantly affected by day (P=.007), but not by treatment or by a day*treatment interaction (Figure 44, Tables A-11 and B-43). The dorsal to palmar ratio increased significantly from day 0 to day 50 (P=.002), decreased from day 50 to day 64 (P=.06), increased slightly from day 64 to day 82, and demonstrated no real change from day 82 to the end of the trial. The Con group had a non-significantly larger ratio of dorsal to palmar RBAE on day 0 than did the eST group. This non-statistical difference between the groups disappeared entirely by day 64 due to a significant decrease in the dorsal to palmar ratio in the Con group from day 52 to day 64 (P=.02).

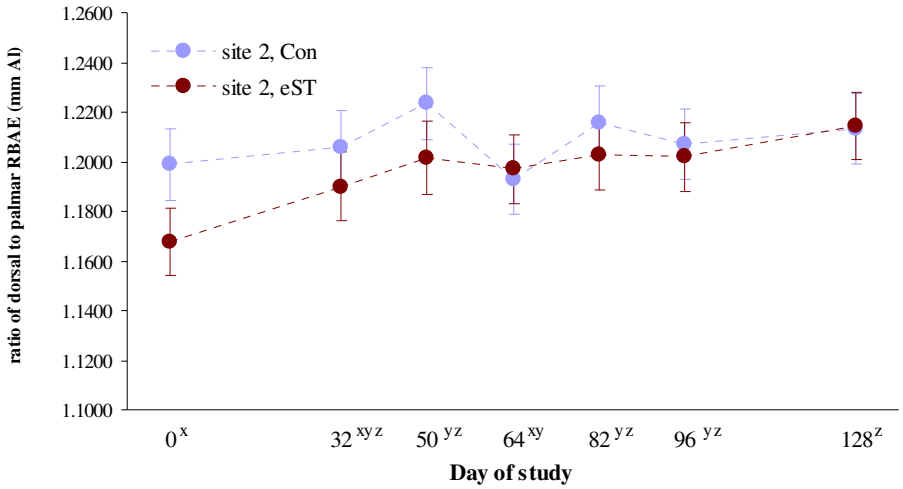


Figure 44. Ratio of dorsal to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 2.

^{xyz} Days not sharing the same superscript differ (P≤.05).

The data of dorsal to palmar RBAE ratio at site 2 were normalized and re-evaluated (Figure 45, Tables A-12 and B-44). Day effects were significant (P=.005), but treatment effects were not of statistical importance. No day*treatment interaction was seen. There was a gain in the normalized ratio from day 0 to day 50 (P=.001), a decrease in the normalized ratio from day 50 to day 64 (P=.04), and a gain in the normalized ratio from day 64 to day 128 (P=.03). The two treatment groups both had a decrease in the normalized ratio from day 50 to day 64. However, the decrease in the eST group was minor and non-significant while the decrease in the Con group was statistically important (P=.01), and resulted in a real difference in the normalized dorsal to palmar RBAE ratio between the two groups on day 64 at site 2 (P=.04). By day 82 there was no longer a statistical difference between the two treatment groups, but there was a trend for the ratio to once again be greater in the eST group than in the Con group by day 128 (P=.06).

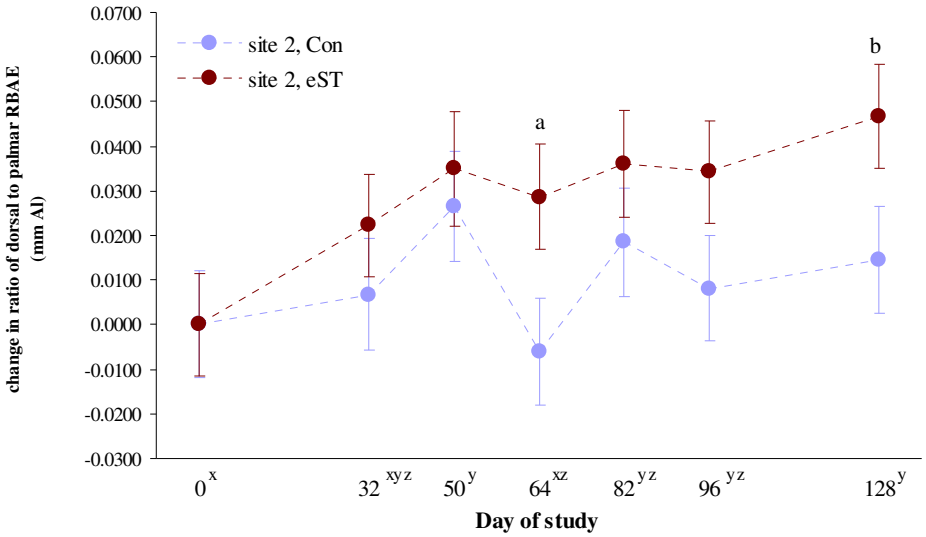


Figure 45. Normalized ratio of dorsal to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 2.

[a] Treatments differ (P≤.05).

[b] Trend for treatments to differ (P≤.10).

[xyz] Days not sharing the same superscript differ (P≤.05).

Site 3

The ratio of dorsal to palmar RBAE at site 3 (Figure 46, Tables A-11 and B-45) was not affected by treatment or by a day*treatment interaction, but there was a significant day affect (P=.004). The increase in the ratio that occurred early in the trial resulted in the dorsal to palmar ratio on day 50 being significantly greater than on day 0 (P=.0007). There was no significant change in the ratio from day 50 to the end of the trial. Though there were minor decreases in the ratio from day 50 to day 64 and from day 82 to day 96, these were offset by minor increases in the ratio from day 64 to day 82 and from day 96 to day 128. The difference seen between the two treatment groups in the ratio of dorsal to palmar RBAE at site 3 on day 0 was not statistically important. Over the course of the project, this difference disappeared entirely.

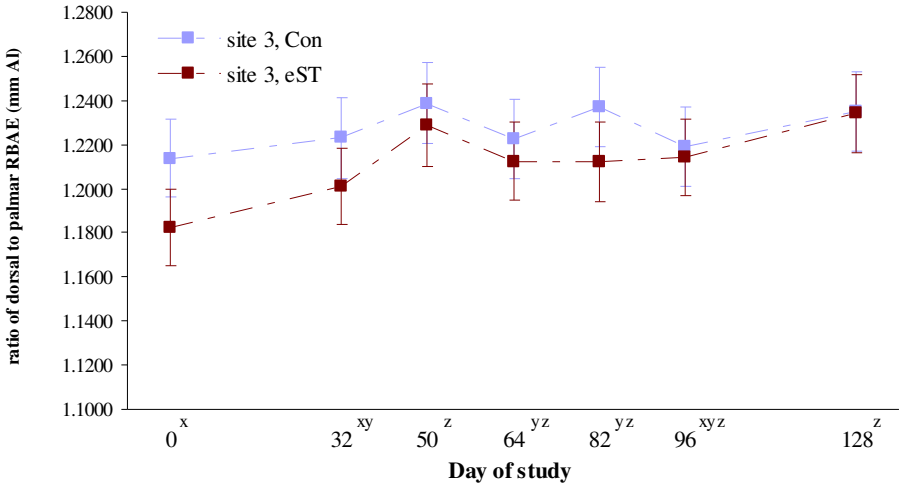


Figure 46. Ratio of dorsal to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 3.

^{xyz} Days not sharing the same superscript differ (P≤.05).

Data from the dorsal to palmar RBAE ratio at site 3 were normalized to day 0 values and re-evaluated (Figure 47, Tables A-12 and B-46). There was no treatment effect or day*treatment interaction. Day effects were significant (P=.003). There was a

significant increase in the dorsal to palmar ratio from day 0 to day 50 (P=.0005). The normalized dorsal to palmar ratio was at the highest level on day 50, decreased non-significantly from day 50 to day 64, had no change from day 64 to day 96, and increased non-significantly to near the day 50 level by day 128. There was a trend for the increase in normalized dorsal to palmar RBAE ratio at site 3 to be greater in the eST group than in the Con group on day 128 (P=.100).

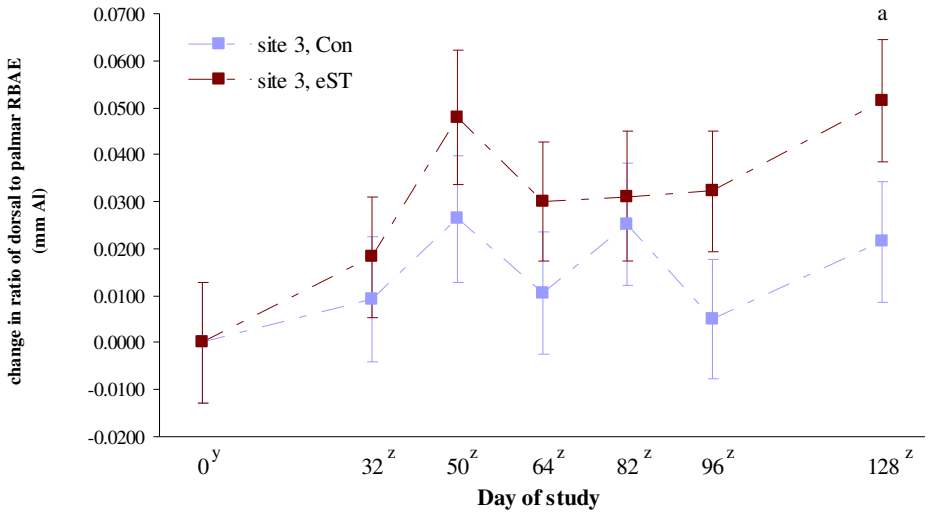


Figure 47. Normalized ratio of dorsal to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 3.

[a] Trend for treatments to differ (P≤.10).

[yz] Days not sharing the same superscript differ (P≤.05).

Site 4

There was not a significant day, treatment, or day*treatment interaction on the ratio of dorsal to palmar RBAE at site 4 (Figure 48, Tables A-11 and B-47), but there was a trend for a day effect (P=.07). The ratio of dorsal to palmar RBAE increased slowly but steadily in the eST group of horses resulting in significant differences from day 0 on day 96 (P=.04) and day 128 (P=.0005). In contrast, there were no significant differences between any of the days measured in the Con group of horses.

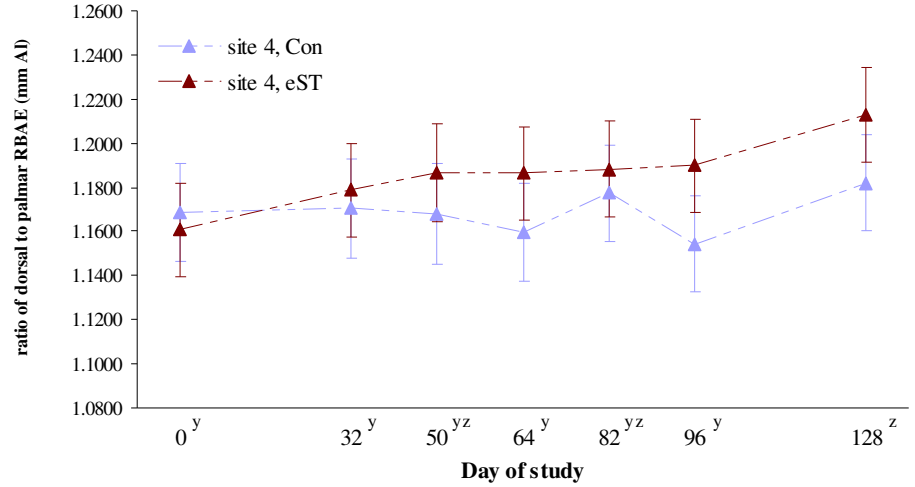


Figure 48. Ratio of dorsal to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 4.

[yz] Days not sharing the same superscript differ (P≤.05).

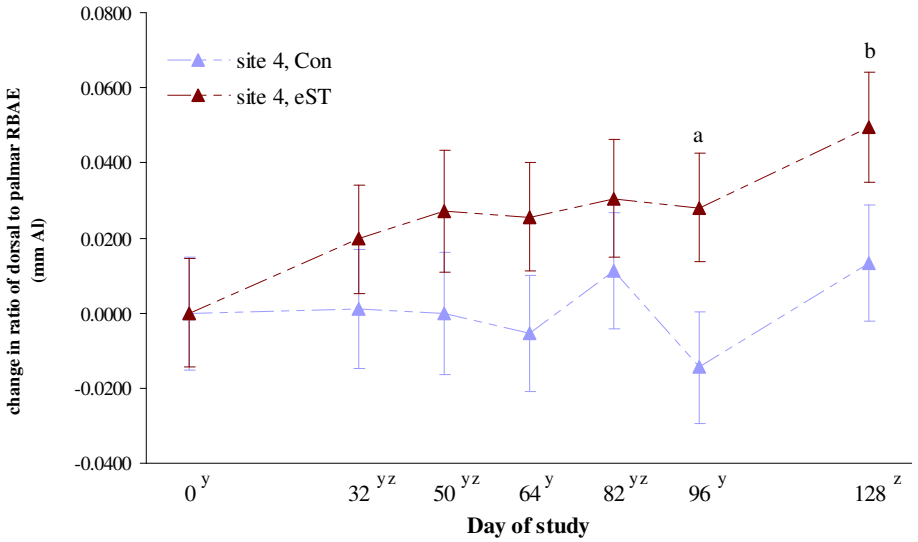


Figure 49. Normalized ratio of dorsal to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 4.

[a] Treatments differ (P≤.05).

[b] Trend for treatments to differ (P≤.10).

[yz] Days not sharing the same superscript differ (P≤.05).

Data from the dorsal to palmar RBAE ratio at site 4 were normalized to day 0 and the statistical analysis were re-run (Figure 49, Tables A-12 and B-48). No statistical differences were seen due to treatment, day, or day*treatment interaction. The normalized ratio increased significantly from day 0 to day 128 (P=.004). The pattern of change in the eST group was a slow but fairly steady gain in the ratio, resulting in the ratio on day 128 being significantly greater than on day 0 (P=.001) or on day 32 (P=.05). In contrast, the Con group had no statistical increase in the ratio during the experiment. The gain in the ratio in the eST group was significantly greater than the ratio in the Con group on day 96 (P=.04), and still had a trend to be greater than the Con group on day 128 (P=.09).

Ratio of Lateral to Palmar RBAE

Site 1

The ratio of lateral to palmar RBAE at site 1 was significantly affected by day (P=.0002), but was not affected by treatment nor by day*treatment interaction (Figure 50, Tables A-13 and B-49). There was a significant increase in the ratio of lateral to palmar RBAE from day 0 to day 32 (P=.002). From day 32 to day 50 there was a decrease in the ratio that showed a trend toward significance (P=.08). The ratio did not change significantly from day 50 to day 96, then increased significantly from day 96 to day 128 (P=.01). The ratio of lateral to palmar RBAE on day 128 was also significantly greater than it had been on day 0 (P<.0001), day 50 (P=.001), day 64 (P=.001), and day 82 (P=.003). The Con group did have a non-statistically greater increase in lateral to palmar RBAE from day 0 to day 32 than the eST group. Additionally, the Con group had a non-significant decrease in the lateral to palmar RBAE ratio from day 82 to day 96 that was not seen in the eST group.

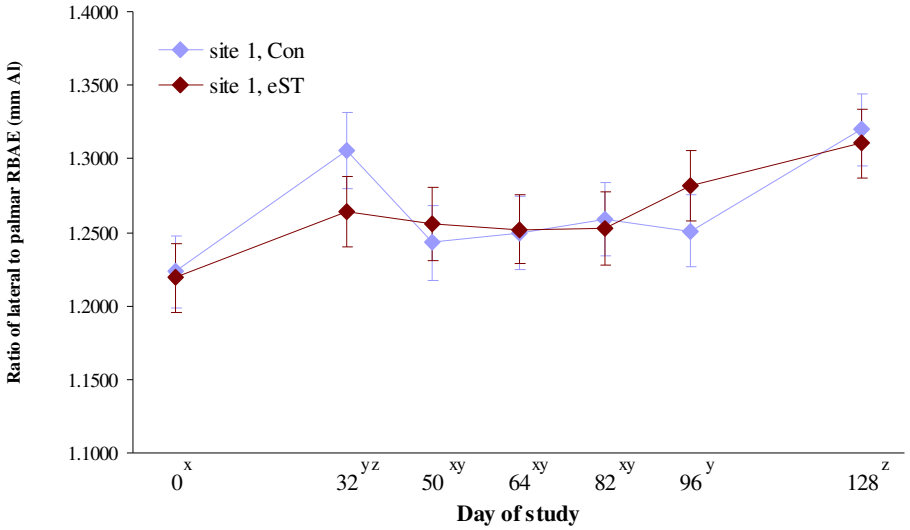


Figure 50. Ratio of lateral to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 1.

[xyz] Days not sharing the same superscript differ (P≤.05).

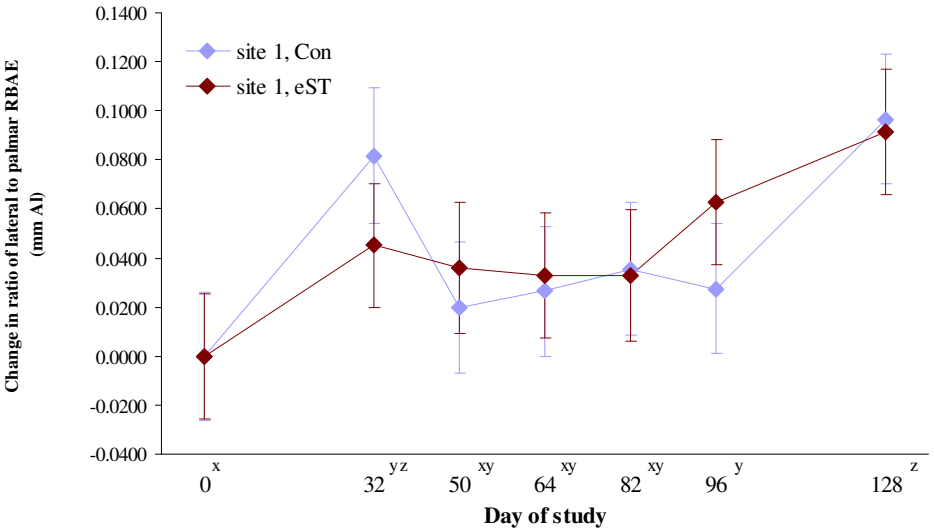


Figure 51. Normalized ratio of lateral to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 1.

[xyz] Days not sharing the same superscript differ (P≤.05).

Data were normalized to day 0 to better observe changes in time in the lateral to palmar RBAE ratio at site 1 (Figure 51, Tables A-14 and B-50). There was a significant day effect (P=.0002) but no treatment effect or day*treatment interaction. There was a significant increase in the ratio from day 0 to day 32 (P=.002). The decrease in the ratio from day 32 to day 50 was not significant. No change was seen in the ratio from day 50 to day 96. The increase in the ratio from day 96 to day 128 resulted in the ratio on day 128 being significantly greater than the ratio on day 0 (P<.0001), day 50 (P=.001), day 64 (P=.001), day 82 (P=.003), and day 96 (P=.01). The two treatment groups did not vary significantly from each other in normalized lateral to palmar RBAE ratio at site 1.

Site 2

The day affects on the ratio of lateral to palmar RBAE at site 2 were significant (P<.0001), but no treatment affects or day*treatment interaction were seen (Figure 52, Tables A-13 and B-51). An increase in the ratio from day 0 to day 32 was significant (P=.001), as was an increase in the ratio from day 96 to day 128 (P=.003). The increase in the ratio was similar between the two treatment groups.

The data were normalized to day 0 values to more closely evaluate changes over time in the lateral to palmar RBAE ratio at site 2 (Figure 53, Tables A-14 and B-52). The normalized data were subjected to statistical analysis. There was not a treatment effect or a day*treatment interaction. There was a significant day effect (P<.0001). A significant gain in the ratio occurred from day 0 to day 32 (P=.001), no change in the ratio occurred from day 32 to day 96, and another significant gain in the ratio occurred from day 96 to day 128 (P=.003). The two treatment groups did not vary significantly from each other in the normalized lateral to palmar RBAE ratio at site 2 on any of the measured days.

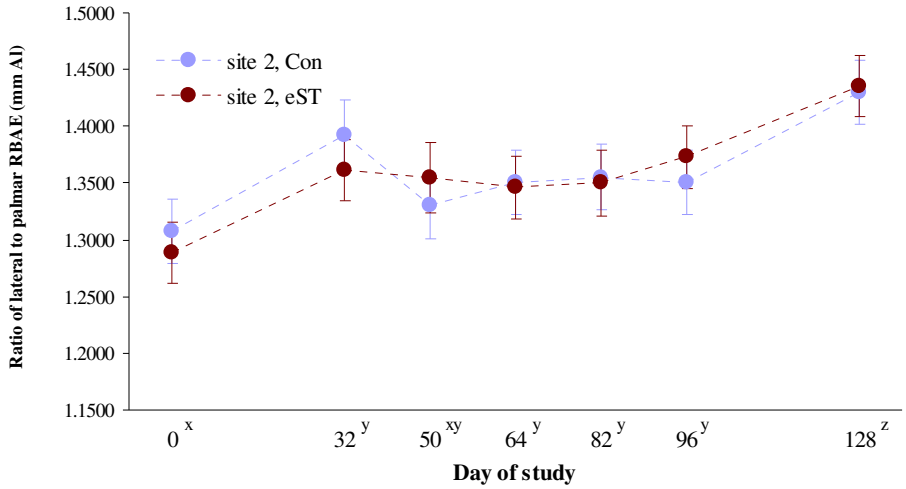


Figure 52. Ratio of lateral to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 2.

[xyz] Days not sharing the same superscript differ (P≤.05).

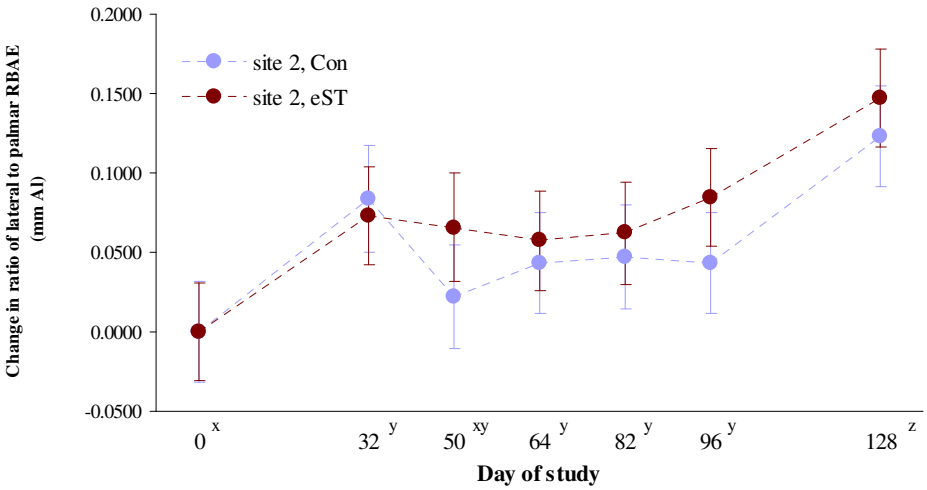


Figure 53. Normalized ratio of lateral to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 2.

[xyz] Days not sharing the same superscript differ (P≤.05).

Site 3

There was a significant day effect on the ratio of lateral to palmar RBAE at site 3 (P<.0001) but there was not a treatment effect or a day*treatment interaction (Figure 54, Tables A-13 and B-53). The lateral to palmar ratio increased significantly from day 0 to day 32 (P=.002). From day 32 to day 50 the ratio had a non-statistical decrease. No change was seen in the ratio from day 50 to day 96. There was a significant increase in the lateral to palmar ratio from day 96 to day 128 (P=.0006). The ratio on day 128 was also significantly greater than the ratio on day 0 (P<.0001), day 32 (P=.007), day 50 (P<.0001), day 64 (P<.0001), and day 82 (P=.0002). The pattern of change in the lateral to palmar RBAE ratio did not differ between the two treatment groups.

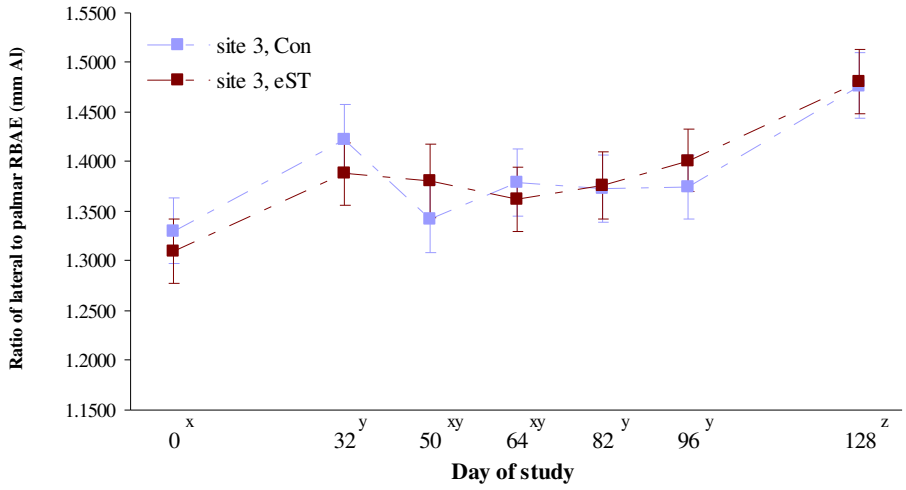


Figure 54. Ratio of lateral to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 3.
^{xyz} Days not sharing the same superscript differ (P≤.05).

Lateral to plamar RBAE ratio data from site 3 were normalized to day 0 values and statistics were re-run to evaluate changes in the ratio over time (Figure 55, Tables A-14 and B-54). Day significantly affected the ratio (P<.0001), but the ratio was not affected by treatment or by any day*treatment interaction. The ratio had a significant gain from

day 0 to day 32 (P=.003), no change from day 32 to day 96, and a significant gain from day 96 to day 128 (P=.0007). The two treatment groups were not significantly different from each other on any of the measured days.

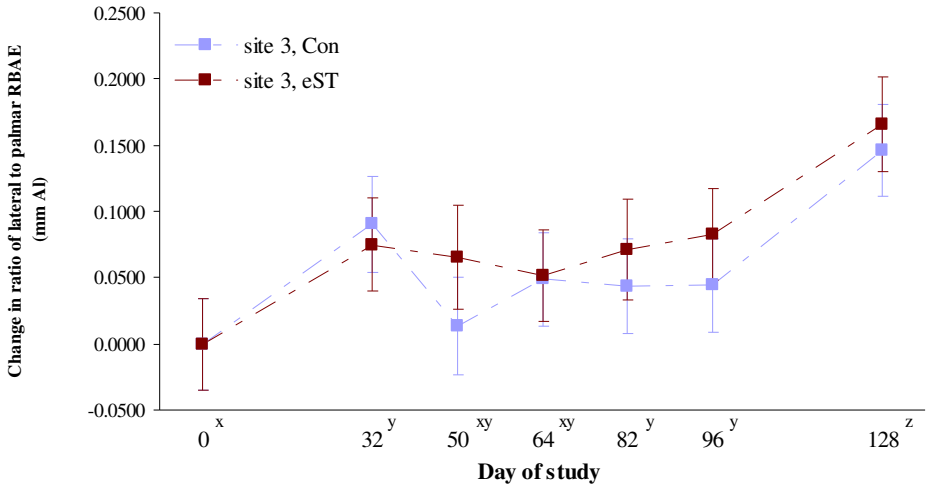


Figure 55. Normalized ratio of lateral to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 3.

^{xyz} Days not sharing the same superscript differ (P≤.05).

Site 4

There were no affects on the ratio of lateral to palmar RBAE at site 4 due to treatment or to day*treatment interaction, but there was a significant day effect (P<.0001) (Figure 56, Tables A-13 and B-55). Similar to what was seen at site1 (Figure 50), site 2 (Figure 52), and site 3 (Figure 54), a significant increase in the ratio of lateral to palmar RBAE occurred from day 0 to day 32 (P=.002), followed by a non-significant decrease from day 32 to day 50, no change from day 50 to day 96, and a significant increase from day 96 to day 128 (P=.0005). The ratio of lateral to plamar RBAE on day 128 was significantly greater than the ratio on day 0 (P<.0001), day 32 (P=.005), day 50 (P<.0001), day 64 (P=.0001), and day 82 (P<.0001). No statistical difference was seen between the two

treatment groups in the pattern of change in the lateral to palmar ratio of RBAE at site 4 over the course of the project.

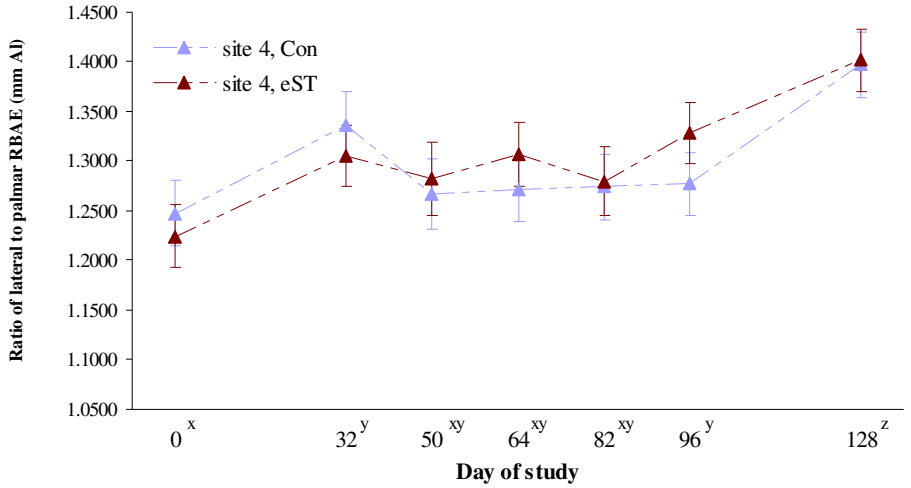


Figure 56. Ratio of lateral to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 4.

^{xyz} Days not sharing the same superscript differ (P≤.05).

The lateral to palmar RBAE ratios at site 4 data were normalized to day 0 values and statistically analyzed (Figure 57, Tables A-14 and B-56). The day effects were significant (P<.0001) but no treatment effects or day*treatment interaction occurred. Similarly to what was seen in the normalized lateral to palmar RBAE ratio data from site 1 (Figure 51), site 2 (Figure 53) and site 3 (Figure 55), there was a significant gain in the ratio from day 0 to day 32 (P=.004), no change from day 32 to day 96, and another significant gain in the ratio from day 96 to day 128 (P=.0006). The two treatment groups did not statistically differ from each other on any given day. There was a non-statistical pattern for the eST group to have a greater normalized lateral to palmar ratio than the Con group on day 50, day 64, day 82, and day 96. A greater gain in the Con group in the ratio from day 96 to day 128 than in the eST group resulted in the two treatment groups being nearly identical in the total change in the lateral to palmar RBAE ratio by day 128.

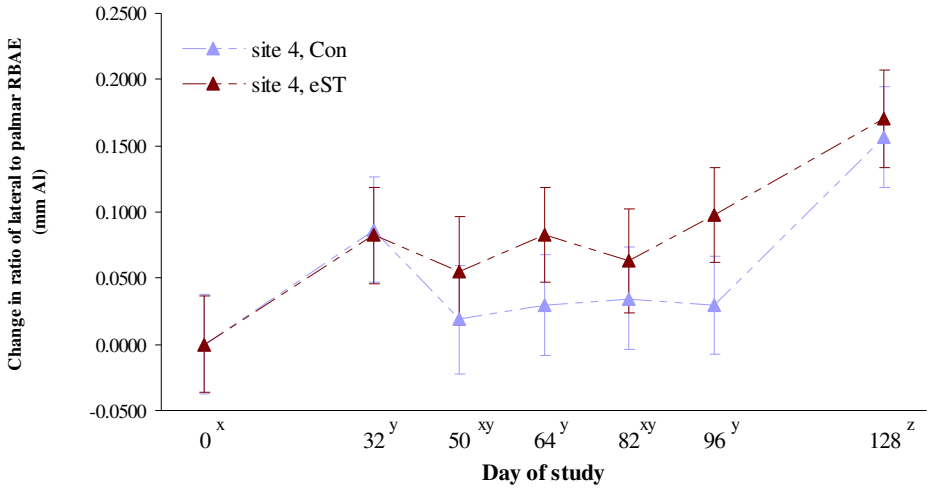


Figure 57. Normalized ratio of lateral to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 4.

^{xyz} Days not sharing the same superscript differ (P≤.05).

Ratio of Medial to Lateral RBAE

Site 1

The ratio of medial to lateral RBAE at site 1 was significantly affected by day (P=.001), but was not affected by treatment or by day*treatment interaction (Figure 58, Tables A-15 and B-57). The ratio increased significantly from day 0 to day 50 (P=.002). No real change in the ratio occurred between day 50 and day 64. A non-significant increase in the medial to lateral ratio of RBAE took place between day 64 and day 82. There was a decrease in the ratio from day 82 to the end of the trial. The medial to lateral RBAE ratio on day 82 was significantly greater than on day 0 (P<.0001), day 32 (P=.003), or day 128 (P=.04). The ratio was non-statistically greater in the Con group on day 0 than in the eST group. This changed by day 32. No difference was seen between the two groups on day 96. The medial to lateral ratio of RBAE was non-significantly greater in the eST group than in the Con group on day 128, a reverse of what was seen on day 0.

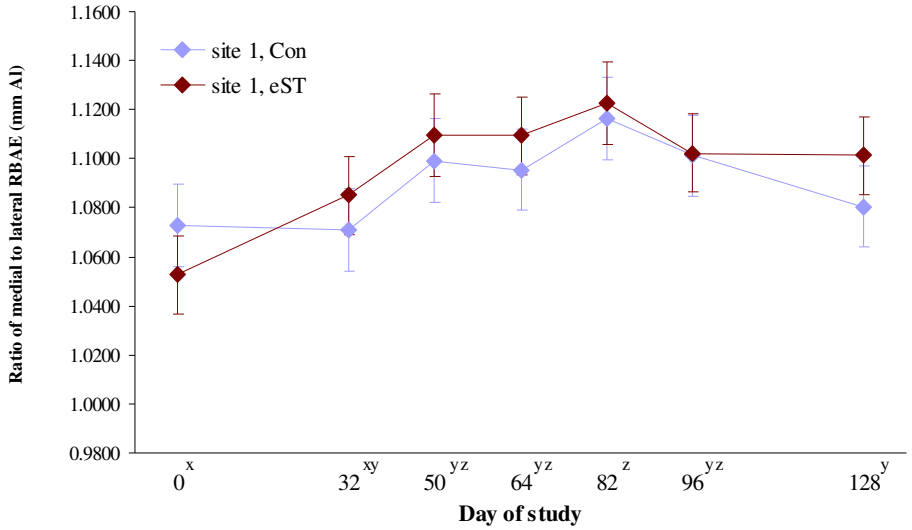


Figure 58. Ratio of medial to lateral radiographic bone aluminum equivalence (RBAE) (mm Al) at site 1.

^{xyz} Days not sharing the same superscript differ (P≤.05).

The data were normalized to day 0 values to better visualize changes in the medial to lateral RBAE ratio at site 1 over time (Figure 59, Tables A-16 and B-58). Day significantly affected the ratio (P=.0008) but treatment did not significantly affect the ratio (P=.101) nor did any day*treatment interaction. There was a significant gain in the ratio from day 0 to day 50 (P=.002). Day 50 values were also significantly greater than those on day 32 (P=.05). There was no change in the ratio from day 50 to day 64. The ratio increased non-significantly from day 64 to day 82. The ratio decreased significantly from day 82 to day 128 (P=.03), but on day 128 the ratio was still significantly greater than on day 0 (P=.03). From day 0 to day 32, the eST group of horses had a trend to increase the normalized medial to lateral RBAE ratio (P=.08). The increase continued to day 50, and resulted in the ratio becoming significantly greater than day 0 by day 50 (P=.002) in the eST group of horses. The Con group of horses did not have a change in the normalized medial to lateral RBAE ratio from day 0 to day 32. This group did have a non-significant increase in the ratio from day 32 to day 50. The difference seen in the normalized ratio between the two groups on day 32 continued throughout the trial, and

by day 128 there was a trend for the eST group to have a greater increase in the medial to lateral RBAE ratio than the Con group (P=.09).

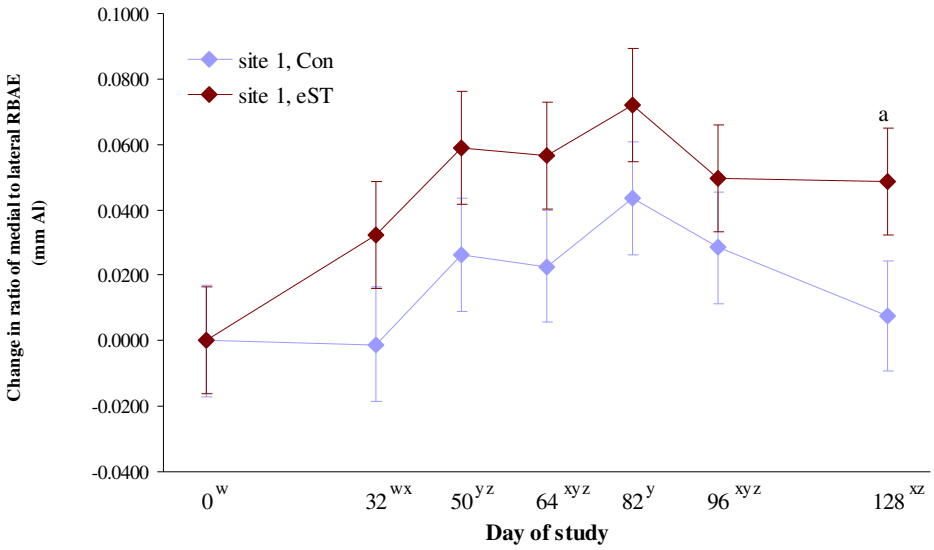


Figure 59. Normailzed ratio of medial to lateral radiographic bone aluminum equivalence (RBAE) (mm Al) at site 1.

[a] Trend for treatments to differ (P≤.10).

[wxyz] Days not sharing the same superscript differ (P≤.05).

Site 2

The ratio of medial to lateral RBAE at site 2 was significantly affected by day (P=.02), but not by treatment or by a day*treatment interaction (Figure 60, Tables A-15 and B-59). The ratio increased slightly from day 0 to day 32, and increased significantly from day 32 to day 50 (P=.02). The ratio on day 50 was also significantly greater than on day 0 (P=.005) and day 128 (P=.04). No real change in the ratio occurred from day 50 to day 82. The ratio of medial to lateral RBAE at site 2 decreased from day 82 to day 128 (P=.05). No real change was observed between the two treatment groups, though the slightly greater ratio seen in the Con group on day 0 was reversed by day 128, with a slightly greater ratio in the eST group than the Con group on that day.

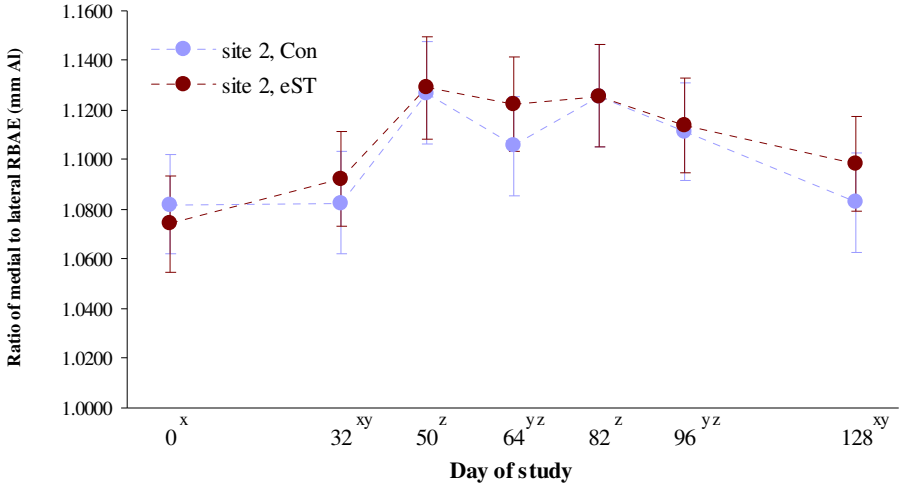


Figure 60. Ratio of medial to lateral radiographic bone aluminum equivalence (RBAE) (mm Al) at site 2.

^{xyz} Days not sharing the same superscript differ (P≤.05).

Treatment effects were not significant, nor was there a day*treatment interaction, but day effects were significant (P=.02) when the medial to lateral RBAE ratio data were normalized to day 0 (Figure 61, Tables A-16 and B-60). The non-significant gain in the ratio from day 0 to day 32 increased from day 32 to day 50. Day 50 medial to lateral RBAE ratio at site 2 was larger than that seen on any other day and was significantly greater than the same ratio on day 0 (P=.004), day 32 (P=.02) and day 128 (P=.03). There was no significant difference in the ratio from day 50 to day 96. A decrease in the ratio from day 96 to day 128 was not significant. The ratio on day 128 was significantly less than on day 50 (P=.03) and day 82 (P=.04), but was not statistically different than the ratio on any of the other days that were sampled. The eST treatment group did gain in the ratio from day 0 to day 32 while the Con group remained unchanged during that same time. However, the two groups did not significantly vary from each other on any day and had a similar pattern of change in the normalized RBAE ratio over the course of the research project.

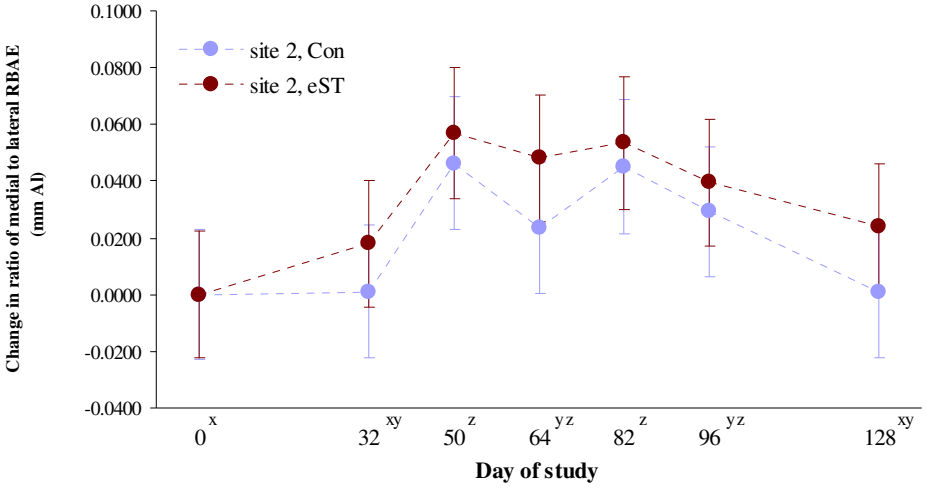


Figure 61. Normailzed ratio of medial to lateral radiographic bone aluminum equivalence (RBAE) (mm Al) at site 2.

^{xyz} Days not sharing the same superscript differ (P≤.05).

Site 3

Day effects were significant (P=.02), but treatment effects and day*treatment interaction were not observed to affect the medial to lateral RBAE ratio at site 3 (Figure 62, Tables A-15 and B-61). The ratio increased significantly from day 0 to day 50 (P=.005), decreased slightly from day 50 to day 82, and decreased significantly from day 82 to day 128 (P=.04). Day 128 medial to lateral ratio of RBAE was not different from the same ratio on day 0. No difference was noted in the two treatment groups until day 64. The day 64 ratio of medial to lateral RBAE decreased in the Con group and remained unchanged in the eST group, though this difference was not statistically important.

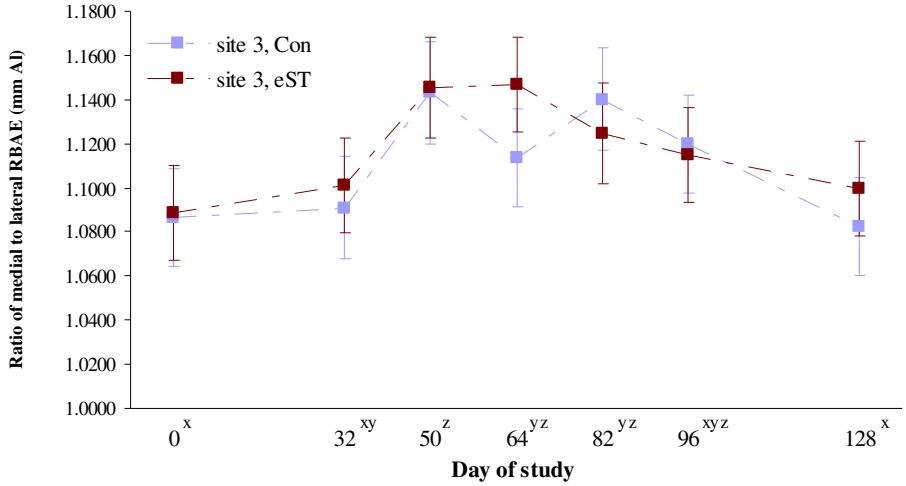


Figure 62. Ratio of medial to lateral radiographic bone aluminum equivalence (RBAE) (mm Al) at site 3.

[xyz] Days not sharing the same superscript differ (P≤.05).

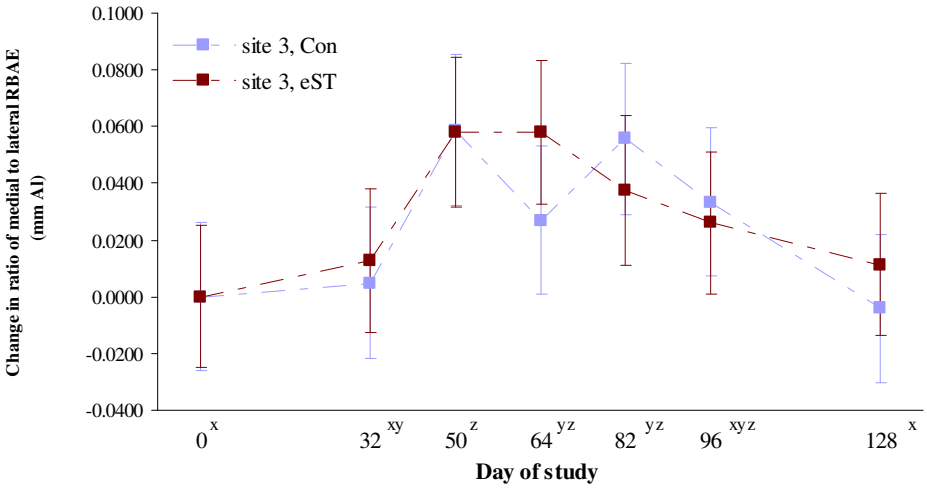


Figure 63. Normalized ratio of medial to lateral radiographic bone aluminum equivalence (RBAE) (mm Al) at site 3.

[xyz] Days not sharing the same superscript differ (P≤.05).

Data were normalized to better observe any changes over time in the medial to lateral RBAE ratio at site 3 (Figure 63, Tables A-16 and B-62). Day effects were significant

(P=.02). There were no effects due to treatment or due to any day*treatment interaction. The ratio did not change from day 0 to day 32, but increased significantly from day 32 to day 50 (P=.01). No statistical change in the ratio occurred from day 50 to day 82. There was a significant decrease in the medial to lateral RBAE ratio from day 82 to day 128 (P=.03). The ratio on day 128 was not significantly different from on day 0. The two treatment groups did not statistically differ from each other on any of the sample days, however the Con group did have a decrease in the ratio on day 64 that did not occur in the eST group.

Site 4

There was not a day*treatment interaction or a treatment effect on the ratio of medial to lateral RBAE at site 4 (Figure 64, Tables A-15 and B-63), but there was a significant day effect (P=.05). The ratio increased significantly from day 0 to day 50 (P=.05). The ratio decreased from day 50 to day 64, and increased from day 64 to day 82, but neither change was significant. The medial to lateral ratio decreased significantly from day 82 to day 128 (P=.007). The ratio on day 128 was below the ratio on day 0, though the two were not statistically different. No differences were appreciated between the two treatment groups.

The data from the medial to lateral RBAE ratio at site 4 were normalized to day 0 and re-evaluted (Figure 65, Tables A-16 and B-64). Day effects were significant (P=.03), but there were still no treatment effects or day*treatment interaction. The ratio increased significantly from day 0 to day 50 (P=.04), with most of the increase occurring between day 32 and day 50. The ratio decreased non-significantly from day 50 to day 64, and increased non-significantly from day 64 to day 82. The ratio decreased significantly from day 82 to day 128 (P=.005). The ratio on day 128 was less than the ratio was on day 0, but the difference between the ratio on day 0 and day 128 was not significant. The pattern of change was not different between the two treatment groups.

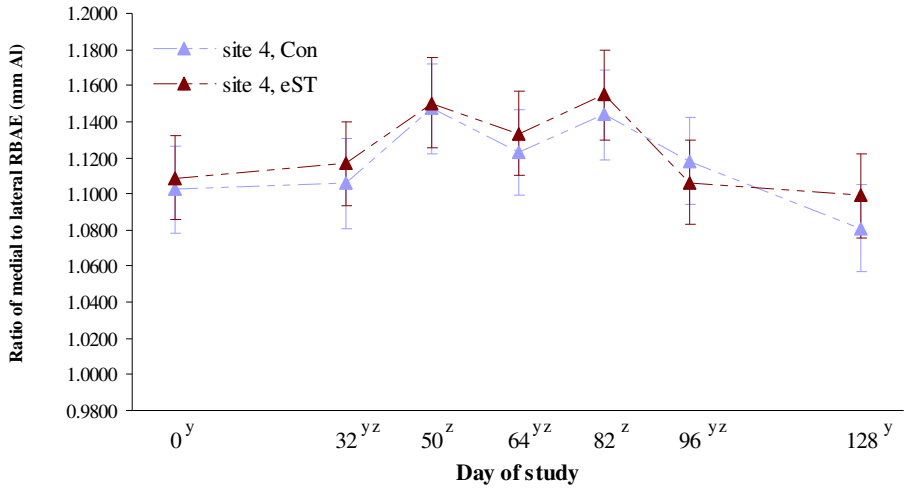


Figure 64. Ratio of medial to lateral radiographic bone aluminum equivalence (RBAE) (mm Al) at site 4.

[yz] Days not sharing the same superscript differ (P≤.05).

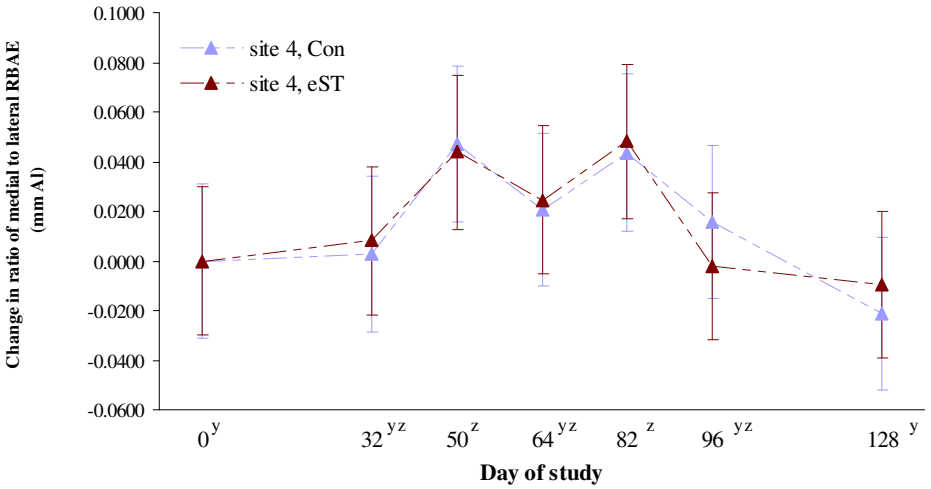


Figure 65. Normalized ratio of medial to lateral radiographic bone aluminum equivalence (RBAE) (mm Al) at site 4.

[yz] Days not sharing the same superscript differ (P≤.05).

Ratio of Medial to Palmar RBAE

Site 1

Day significantly affected the ratio of medial to lateral RBAE at site 1 (P<.0001), but treatment did not affect the ratio, nor did day*treatment interaction (Figure 66, Tables A-17 and B-65). The ratio increased significantly from day 0 to day 32 (P<.0001), increased non-significantly from day 32 to day 96, and once again increased significantly from day 96 to day 128 (P=.03). No differences were seen between the two treatment groups.

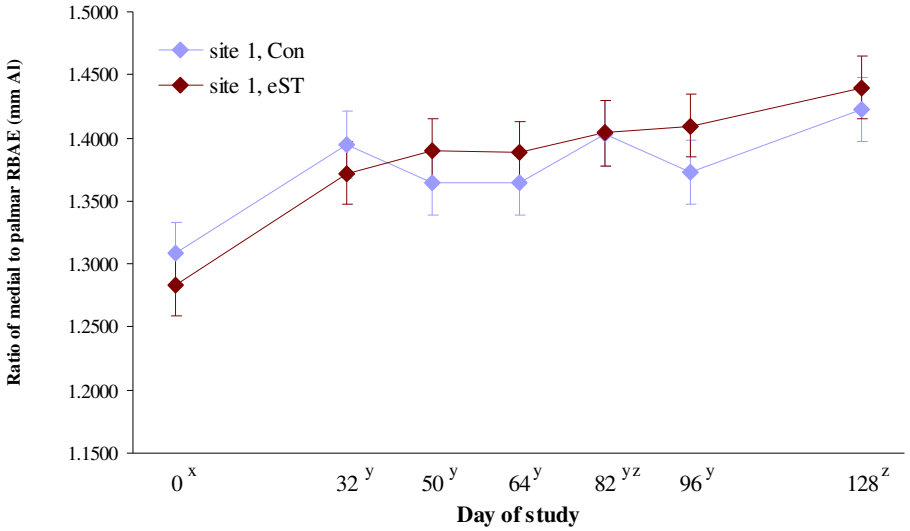


Figure 66. Ratio of medial to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 1.

^{xyz} Days not sharing the same superscript differ (P≤.05).

The data were normalized to day 0 to better evaluate changes in the medial to palmar RBAE ratio at site 1 over time (Figure 67, Tables A-18 and B-66). Day effects were significant (P<.0001), but there were no significant effects due to treatment or to day*treatment interaction. There was a significant increase in the ratio from day 0 to day 32 (P<.0001), no change from day 32 to day 96, and a second significant increase in the

ratio from day 96 to day 128 (P=.03). The Con group had a non-significant decrease in the ratio from day 32 to day 50. In contrast, the eST group had a non-significant increase in the ratio from day 32 to day 50. Though never significant, the difference in gain in the medial to palmar RBAE ratio that occurred between the two treatment groups as a result of the difference in gain from day 32 to day 50 continued through the end of the trial, with a greater increase in the ratio in the eST group than in the Con group.

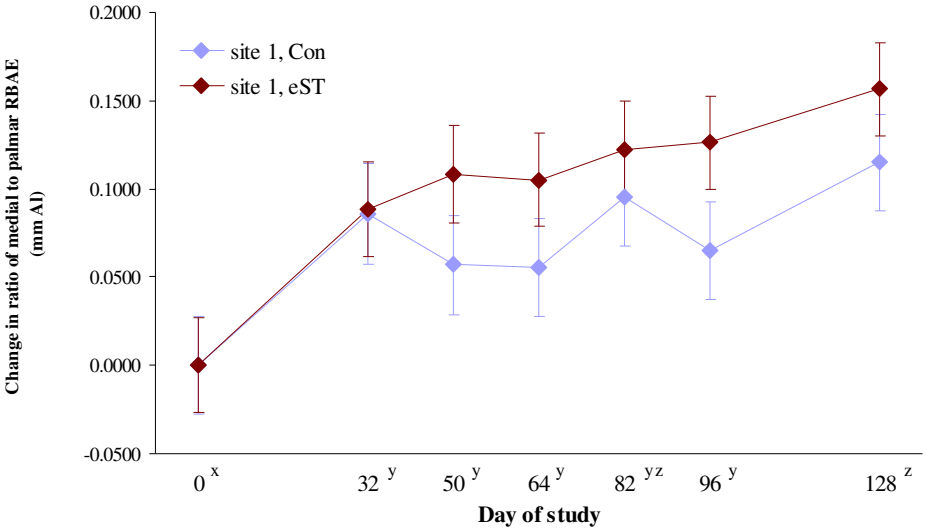


Figure 67. Normalized ratio of medial to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 1.

[xyz] Days not sharing the same superscript differ (P≤.05).

Site 2

The ratio of medial to palmar RBAE at site 2 was significantly affected by day (P<.0001), but not by treatment or by day*treatment interaction (Figure 68, Tables A-17 and B-67). The pattern of change in the medial to palmar RBAE ratio at site 2 was the similar to that seen at site 1 (Figure 66). A significant increase in the ratio occurred from day 0 to day 32 (P<.0001), no real change in the ratio occurred from day 32 to day 96,

and a second significant increase in the ratio was seen from day 96 to day 128 (P=.02). No difference in the ratio was seen between the two treatment groups at site 2.

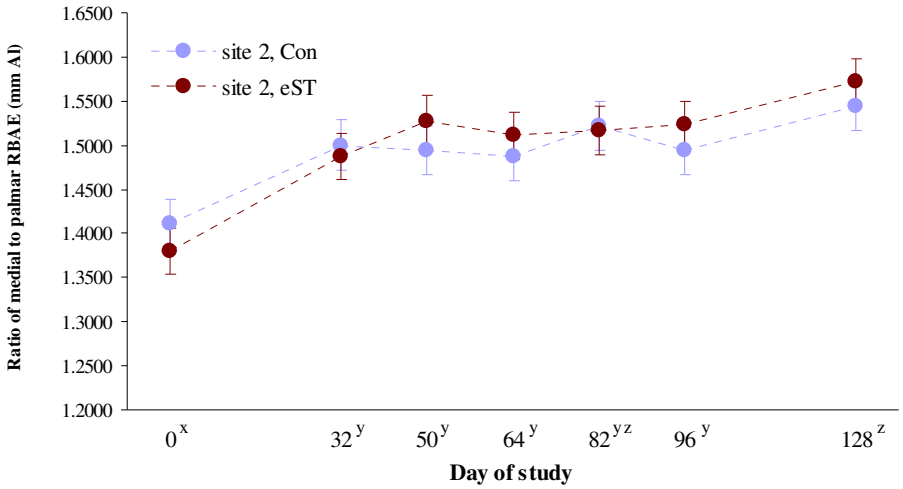


Figure 68. Ratio of medial to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 2.

[xyz] Days not sharing the same superscript differ (P≤.05).

The medial to palmar RBAE ratio data from site 2 were normalized to day 0 and statistical analysis were run on the normalized data (Figure 69, Tables A-18 and B-68). Treatment effects were not significant, nor was there a day*treatment interaction. Day effects were significant (P<.0001). An increase in the normalized medial to palmar RBAE ratio from day 0 to day 32 was significant (P<.0001). There was no change in the ratio from day 32 to day 96, and then a significant increase in the ratio from day 96 to day 128 (P=.02). There was not a significant difference between the two treatment groups though the eST group had an increase in the ratio from day 32 to day 50 that was not seen in the control group. The resulting greater increase in the ratio in the eST group than in the Con group continued through day 128 but never reached statistical importance.

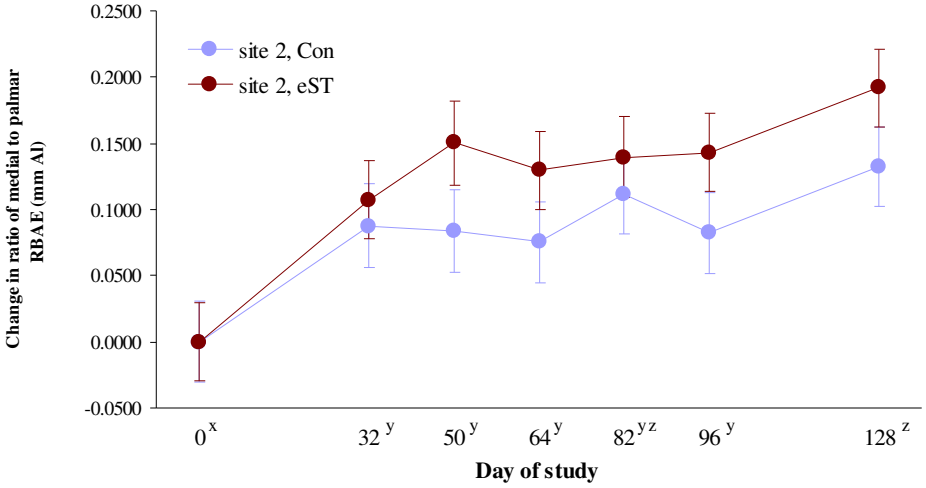


Figure 69. Normalized ratio of medial to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 2.

^{xyz} Days not sharing the same superscript differ (P≤.05).

Site 3

The ratio of medial to palmar RBAE at site 3 was significantly affected by day (P<.0001), but was not affected by treatment, nor by a day*treatment interaction (Figure 70, Tables A-17 and B-69). The pattern of change in the ratio was similar to that seen at site 1 (Figure 66) and site 2 (Figure 68). There was an increase in the medial to palmar ratio from day 0 to day 32 (P<.0001). No statistical change in the ratio occurred from day 32 to day 96. The ratio increased significantly from day 96 to day 128 (P=.007). The two treatment groups had the same pattern of change in the ratio of medial to palmar RBAE at site 3.

Data from the ratio of medial to palmar RBAE at site 3 were compared to day 0 values and the resulting normalized data were subjected to statistical analysis (Figure 71, Tables A-18 and B-70). Day effects were significant (P<.0001), but treatment effects were not significant, nor was there a day*treatment interaction. An increase in the ratio from day 0 to day 32 was significant (P<.0001), as was an increase in the ratio from day 96 to day 128 (P=.008). There was no significant difference in the ratio from day 32 to

day 96. There was no significant difference between the two treatment groups on any of the observed days.

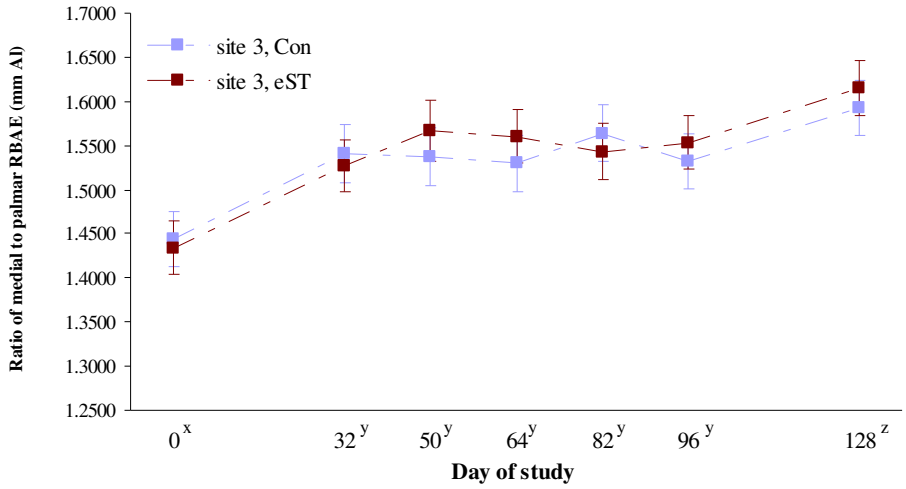


Figure 70. Ratio of medial to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 3.

[xyz] Days not sharing the same superscript differ (P≤.05).

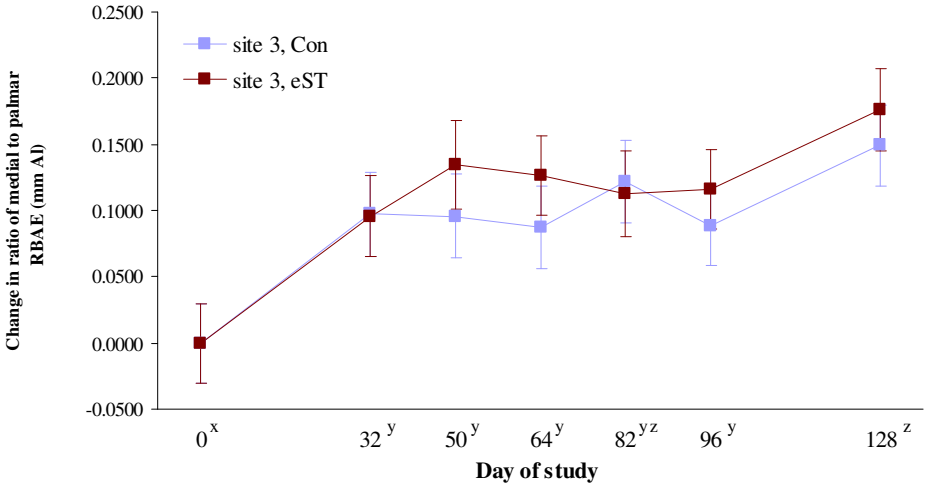


Figure 71. Normalized ratio of medial to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 3.

[xyz] Days not sharing the same superscript differ (P≤.05).

Site 4

Day effects on the ratio of medial to palmar RBAE at site 4 were significant (P<.0001) but treatment effects were not significant, nor was there a day*treatment interaction (Figure 72, Tables A-17 and B-71). The overall pattern of increase in the medial to palmar RBAE ratio was similar to that seen at site 1 (Figure 66), site 2 (Figure 68) and site 3 (Figure 70). There was an increase in the ratio from day 0 to day 32 (P=.0004). There was not a significant change in the ratio from day 32 to day 96. There was a significant increase in the ratio from day 96 to day 128 (P=.002). There was no statistical difference between the two treatment groups in the medial to palmar RBAE ratio at site 4. There was, however, a decrease in the ratio on day 64 in the Con group that did not occur in the eST group.

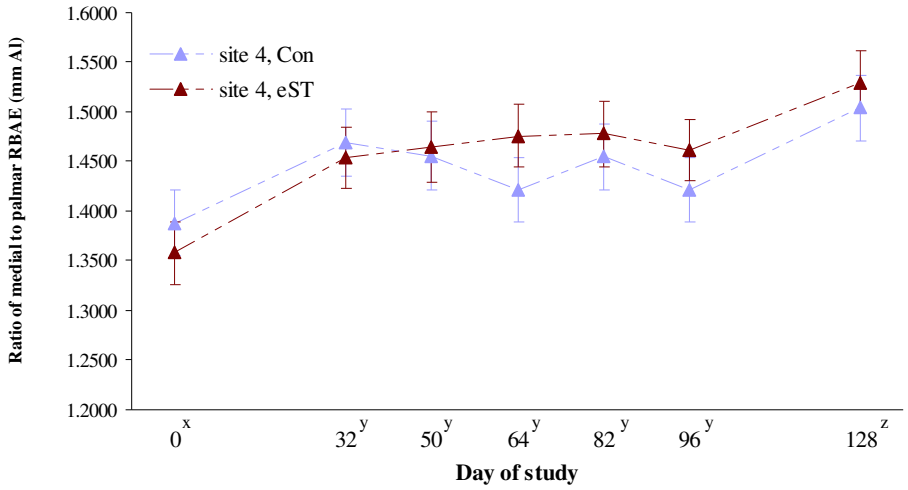


Figure 72. Ratio of medial to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 4.

[xyz] Days not sharing the same superscript differ (P≤.05).

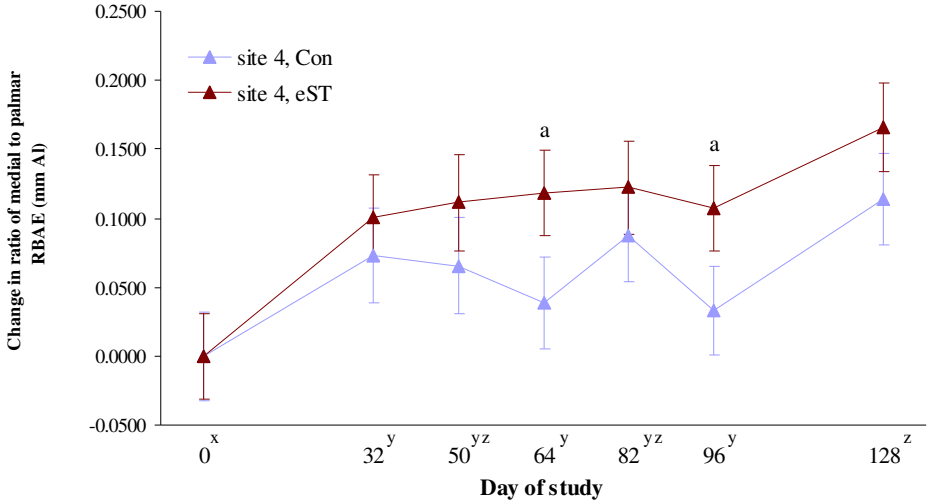


Figure 73. Normalized ratio of medial to palmar radiographic bone aluminum equivalence (RBAE) (mm Al) at site 4.

[a] Trend for treatments to differ (P≤.10).

[xyz] Days not sharing the same superscript differ (P≤.05).

The medial to palmar RBAE ratio data from site 4 were normalized to day 0 values and statistics were re-run (Figure 73, Tables A-18 and B-72). Day had significant affects on the ratio (P<.0001), but treatment did not have an affect on the ratio, nor did day*treatment interaction. The normalized medial to lateral ratio at site 4 increased significantly from day 0 to day 32 (P=.0005), did not change from day 32 to day 96, and increased significantly from day 96 to day 128 (P=.005). The two treatment groups began to differ from each other, though not statistically, on day 32. The eST group had a greater increase in the ratio from day 0 to day 32 than did the Con group. From day 32 to day 82, the eST group continued to increase the normalized medial to palmar ratio, though not significantly. From day 32 to day 64, the Con group, in direct contrast, had a non-significant decrease in the ratio. By day 64, the difference in the ratio between the two treatment groups had a trend to differ (P=.08). The Con group had a non-significant increase in the ratio from day 64 to day 82. Both treatment groups had a non-significant decrease in the ratio from day 82 to day 96. The decrease from day 82 to day 96 was greater in the Con group than in the eST group, causing a trend for the two groups to

differ on day 96 (P=.100). There was no statistical difference between the two treatment groups on day 128.

Dorsal Cortical Bone Width Micrometer Readings

Site 1

There was a significant day effect (P<.0001) and a significant day*treatment interaction (P=.03), but not a significant treatment effect on the dorsal cortical bone width micrometer readings at site 1 (Figure 74, Tables A-19 and B-73). There was a non-significant increase in the width of the dorsal cortical bone from day 0 to day 64, and a significant increase in the width from day 64 to day 128 (P<.0001). The eST group of horses started the trial with a dorsal cortical bone at site 1 that was less thick than the dorsal cortical bone in the Con group of horses, and this continued through day 64. From day 64 to day 128 the eST group had a greater gain in the thickness of the dorsal cortical bone than did the Con group. Though the difference between the two groups was not significant, the greater gain in the eST group allowed that group to finish the trial with greater dorsal cortical bone thickness than did the Con group.

The data from the micrometer readings of the dorsal cortical bone width at site 1 were normalized to day 0 and re-evaluated to better observe the changes in the bone width over time (Figure 75, Tables A-20 and B-74). There was a significant day effect (P<.0001) and a significant day*treatment interaction (P=.03), but not a significant treatment effect on the normalized dorsal cortical bone micrometer readings at site 1. There was a non-significant increase in the width of the dorsal cortical bone from day 0 to day 64, and a significant increase in the width from day 64 to day 128 (P<.0001). There was no difference between the two treatment groups on day 64. The eST group had a significant increase in the micrometer reading from day 64 to day 128 (P<.0001), while the Con group had a trend for an increase in the micrometer reading to be significant during the same time period (P=.09). This resulted in a significantly greater increase in the dorsal cortical width in the eST group of horses as compared to the Con group of horses on day 128 (P=.003).

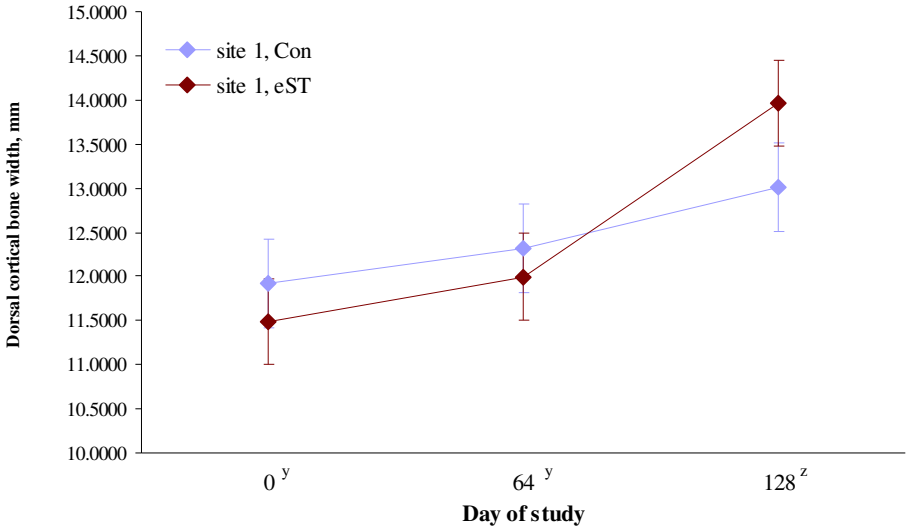


Figure 74. Dorsal cortical bone width in mm, at site 1, vs day of study.

[yz] Days not sharing the same superscript differ (P≤.01).

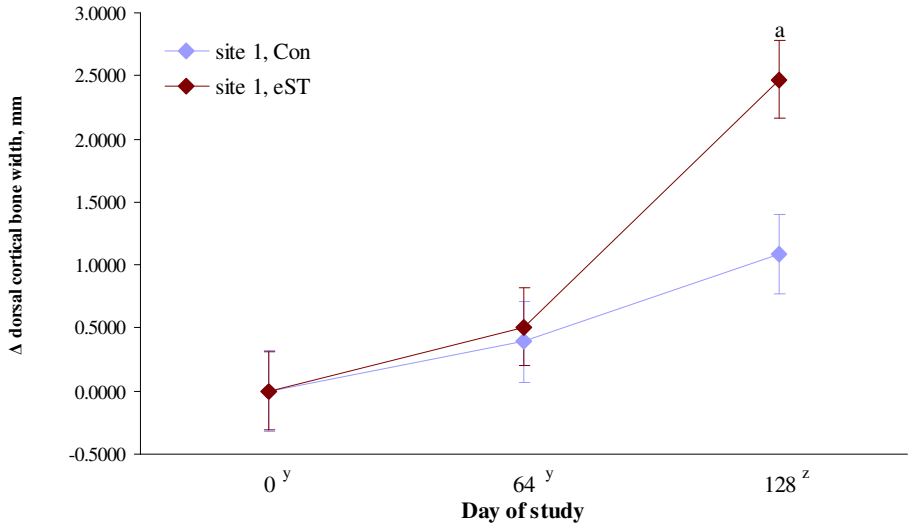


Figure 75. Change in dorsal cortical bone width in mm, at site 1, vs day of study.

[a] Treatments differ (P≤.01).

[yz] Days not sharing the same superscript differ (P≤.01).

Site 2

The dorsal cortical bone width at site 2 was significantly affected by day (P<.0001), and by a day*treatment interaction (P=.02), but not by treatment (Figure 76, Tables A-19 and B-75). No significant difference in the dorsal cortical bone width occurred from day 0 to day 64. There was a significant increase in the dorsal bone width from day 64 to day 128 (P<.0001). The Con group started the project with a non-statistical greater dorsal cortical bone width than the eST group, and this continued to day 64. Between day 64 and day 128 this pattern was reversed, with a greater, but not statistically different, dorsal cortical bone thickness at site 2 in the eST group on day 128 than in the Con group.

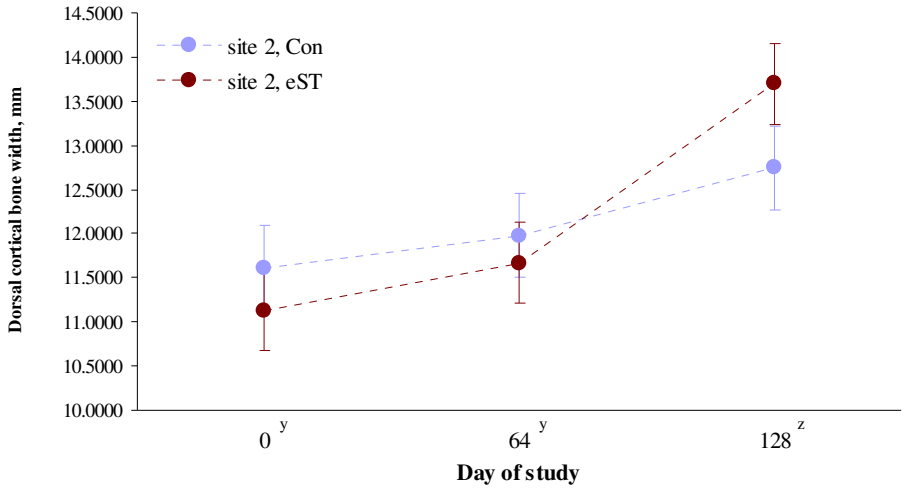


Figure 76. Dorsal cortical bone width in mm, at site 2, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.01).

The data of the dorsal cortical bone width at site 2 were adjusted to remove day 0 values from the values at day 64 and day 128 (Figure 77, Tables A-20 and B-76). When the resulting data were evaluated significant effects were seen due to day (P<.0001) and due to day*treatment interaction (P=.09). A trend for a treatment effect was also seen (P=.09). An increase in dorsal cortical bone width at site 2 was not significant from day

0 to day 64, but was significant (P<.0001) from day 64 to day 128. The treatments did not differ from each other on day 64 but did differ from each other on day 128 (P=.002) due to a greater increase in dorsal cortical bone width in the eST group than in the Con group from day 64 to day 128.

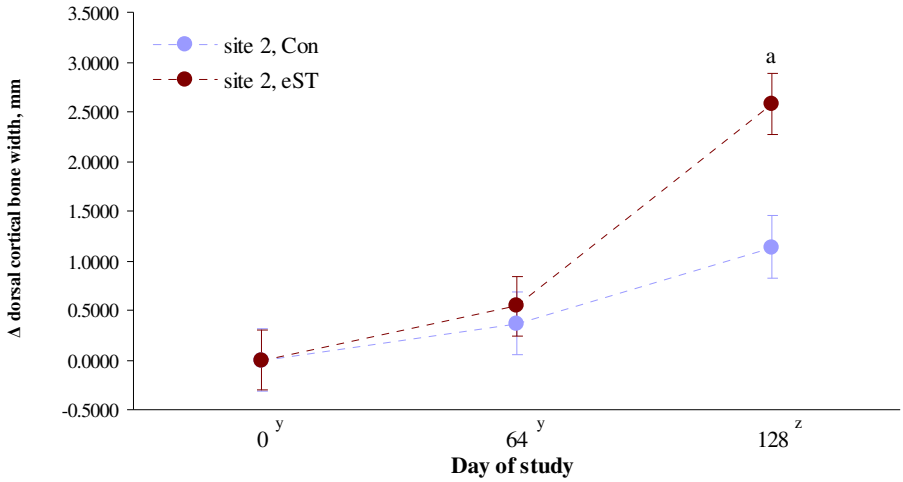


Figure 77. Change in dorsal cortical bone width in mm, at site 2, vs day of study.

[a] Treatments differ (P≤.05).

[yz] Days not sharing the same superscript differ (P≤.01).

Site 3

There were significant day effects (P<.0001) and a day*treatment interaction (P=.03), but no significant treatment effects on the dorsal cortical bone width at site 3 (Figure 78, Tables A-19 and B-77). There was a gain in the cortical bone width in the dorsal cortex at site 3 that reached significance from day 64 to day 128 (P<.0001). The increase from day 64 to day 128 was greater in the eST group of horses than in the Con group of horses, though this difference was not significant.

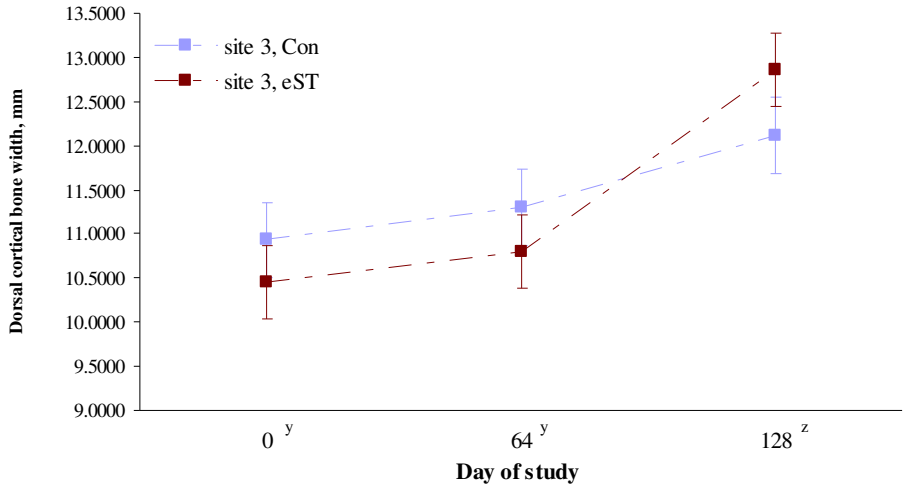


Figure 78. Dorsal cortical bone width in mm, at site 3, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.01).

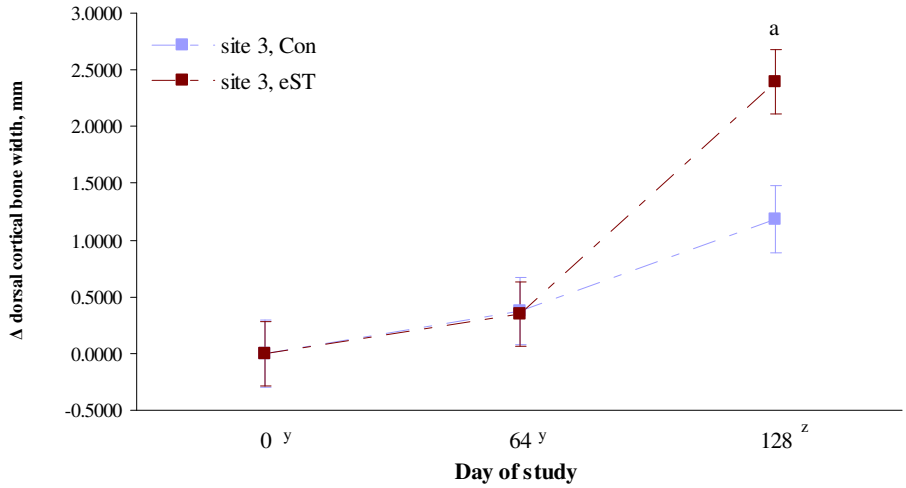


Figure 79. Change in dorsal cortical bone width in mm, at site 3, vs day of study.
[a] Treatments differ (P≤.01).
[yz] Days not sharing the same superscript differ (P≤.01).

The data were normalized to better evaluate any significant changes between the two treatment groups in dorsal cortical bone width at site 3 over time (Figure 79, Tables A-20

and B-78). Statistical ananlysis showed significant effects due to day (P<.0001), and due to a day*treatment interaction (P=.03), but not due to treatment. There was an increase in the normalized dorsal cortical bone width that became significant from day 64 to day 128 (P<.0001). The two treatment groups did not differ from each other until after day 64. From day 64 to day 128, the eST group of horses had a greater gain in dorsal cortical bone width at site 3 than did the Con group, thus the two groups differed from each other on day 128 (P=.004).

Site 4

There were no treatment effects or day*treatment interactions that affected the dorsal cortical bone width at site 4, but day effects were significant (P<.0001) (Figure 80, Tables A-19 and B-79). There were no significant changes in cortical width from day 0 to day 64. Between day 64 and day 128 a significant (P<.0001) increase in the dorsal cortical width occurred. No statistical difference occurred between the two treatment groups in the dorsal cortical width over the course of the experiment.

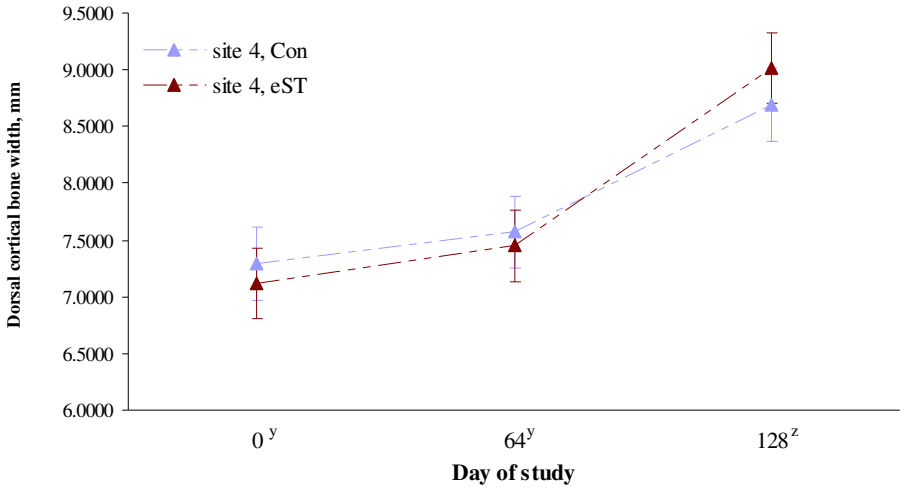


Figure 80. Dorsal cortical bone width in mm, at site 4, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.01).

The dorsal cortical width data from site 4 were normalized to day 0 values and re-evaluated (Figure 81, Tables A-20 and B-80). Day effects were significant (P<.0001), but there was not a treatment effect or a day*treatment interaction. The non-significant increase in dorsal cortical bone width from day 0 to day 64 increased in magnitude and was significant (P<.0001) from day 64 to day 128. The two treatment groups did not differ from each other on day 64. A greater increase in dorsal cortical bone width at site 4 in the eST group than in the Con group from day 64 to day 128 caused the two treatments to have a trend to differ from each other on day 128 (P=.08).

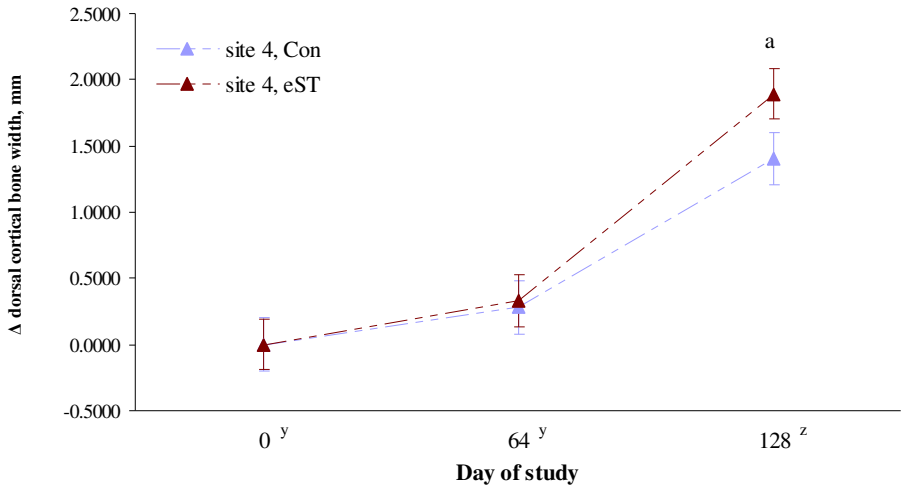


Figure 81. Change in dorsal cortical bone width in mm, at site 4, vs day of study.

[a] Trend for treatments to differ (P≤.10).

[yz] Days not sharing the same superscript differ (P≤.01).

Lateral Cortical Bone Width Micrometer Readings

Site 1

There were no significant effects on the lateral cortical bone width at site 1 due to treatment, day, or day*treatment interaction (Figure 82, Tables A-21 and B-81). No significant changes occurred in the width of the lateral cortical cortex at site 1 over the course of the trial, though there was a trend for the increase from day 0 to day 64 to be

important (P=.06). There was no change over the experiment in the lateral cortical width at site 1 in the eST group of horses. The Con group of horses, however, had a trend for the increase in lateral cortical bone width from day 0 to day 64 to be significant (P=.07).

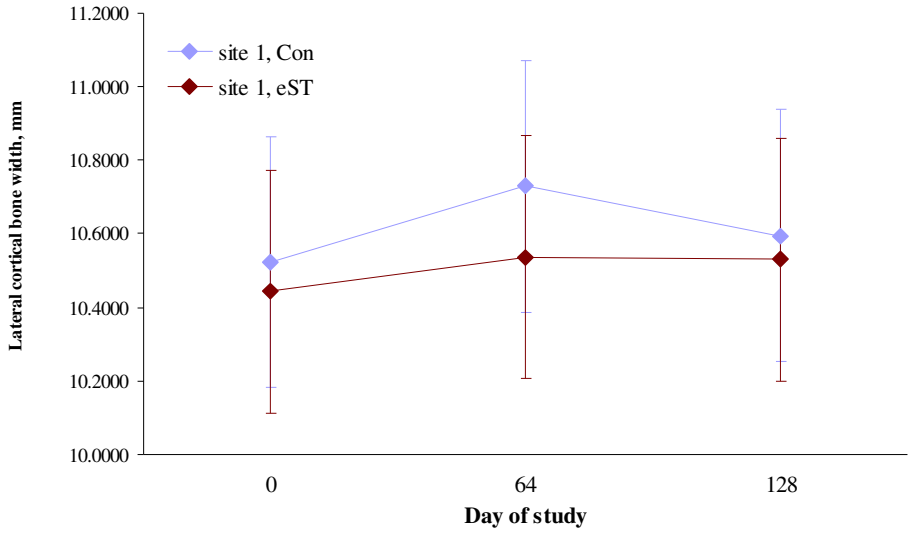


Figure 82. Lateral cortical bone width in mm, at site 1, vs day of study.

The data from the lateral cortical bone width at site 1 were normalized to day 0 values to better evaluate changes over time and statistical analysis was re-run (Figure 83, Tables A-22 and B-81). Once again, at site 1 there was not a treatment, day, or day*treatment interaction that significantly affected the lateral cortical bone width during this project. There was a trend for an increase in cortical width from day 0 to day 64 (P=.06) due to a non-statistical (P=.07) increase in the lateral cortex of the Con group of horses. There were no significant changes in the lateral cortical bone width of eST group of horses during the research project.

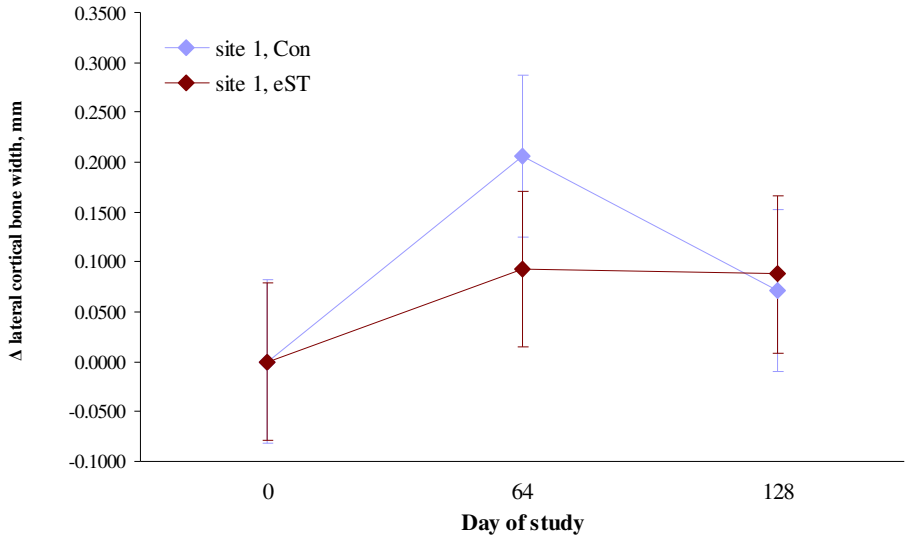


Figure 83. Change in lateral cortical bone width in mm, at site 1, vs day of study.

Site 2

The lateral cortical bone width at site 2 was significantly affected by day (P=.04), but not by treatment or by any day*treatment interaction (Figure 84, Tables A-21 and B-83). The day 64 measured cortical width was significantly greater than that measured on day 0 (P=.02) and on day 128 (P=.05). The Con group of horses started the trial with a non-significantly greater width of the lateral cortex at site 2 than the eST group of horses, and this continued through day 128.

The data from the lateral cortical bone width at site 2 were normalized by subtracting the day 0 values from each measurement to better evaluate changes in the bone width over the course of the experiment. The resulting normalized data were statistically analysed (Figure 85, Tables A-22 and B-84). The normalized lateral cortical bone width at site 2 was significantly affected by day (P=.04), but not by treatment or by a day*treatment interaction. There was a significant increase in the width of the lateral cortex from day 0 to day 64 (P=.02), followed by a significant decrease from day 64 to day 128 (P=.05). From day 64 to day 128, the eST group of horses had a greater

decrease in the width of the lateral cortical bone than did the Con group of horses, but this difference was not of statistical importance.

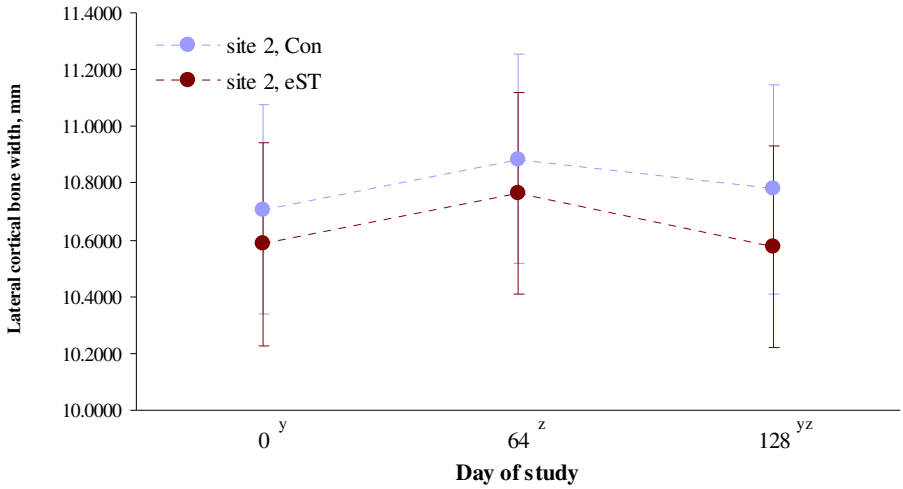


Figure 84. Lateral cortical bone width in mm, at site 2, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.05).

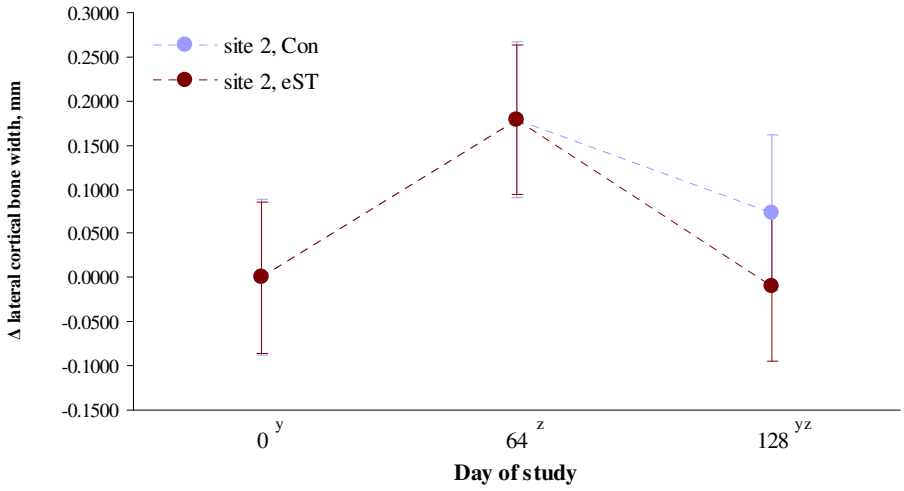


Figure 85. Change in lateral cortical bone width in mm, at site 2, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.05).

Site 3

There was a trend for day effects (P=.07), but no treatment or day*treatment interaction that affected the lateral cortical bone width at site 3 (Figure 86, Tables A-21 and B-85). There was a significant increase in the width of the lateral cortex at site 3 from day 0 to day 64 (P=.03) followed by a trend for a decrease in the cortical width from day 64 to day 128 (P=.07). On day 0 the Con group had a non-statistically greater width in the lateral cortical bone at site 3 than did the eST group. This was gone by day 128.

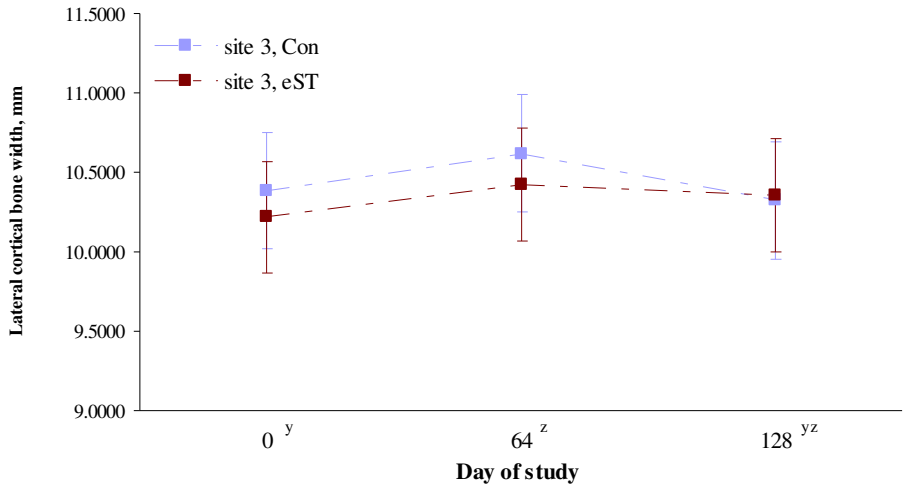


Figure 86. Lateral cortical bone width in mm, at site 3, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.05).

The data from the lateral cortical bone width at site 3 were normalized to day 0 values and re-evaluated (Figure 87, Tables A-22 and B-86). There was a trend for day affects (P=.07), but no treatment or day*treatment affects on the width of the lateral cortex at site 3. The width of the lateral cortical bone increased significantly (P=.03) from day 0 to day 64, and then had a trend to decrease (P=.07) from day 64 to day 128. The change in the lateral cortical width was the same in both the treatment groups through day 64,

but the Con group had a greater loss in lateral cortical width at site 3 from day 64 to day 128 than did the eST group. Thus, there was a trend (P=.09) for the eST group to have a greater increase in lateral cortical bone on day 128 than did the Con group.

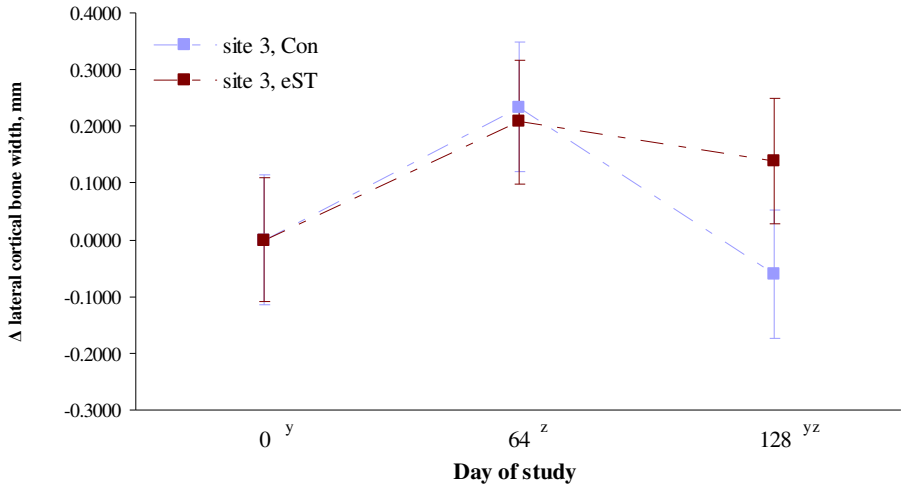


Figure 87. Change in lateral cortical bone width in mm, at site 3, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.05).

Site 4

There were no treatment, day, or day*treatment effects on the lateral cortical bone width at site 4 (Figure 88, Tables A-21 and B-87). No significant differences in the width of the lateral cortex at site 4 occurred during the research trial. The Con group of horses did start the project with a non-significantly greater width of the lateral cortex than the eST group, and this difference increased, but did not reach significance during the duration of the experiment.

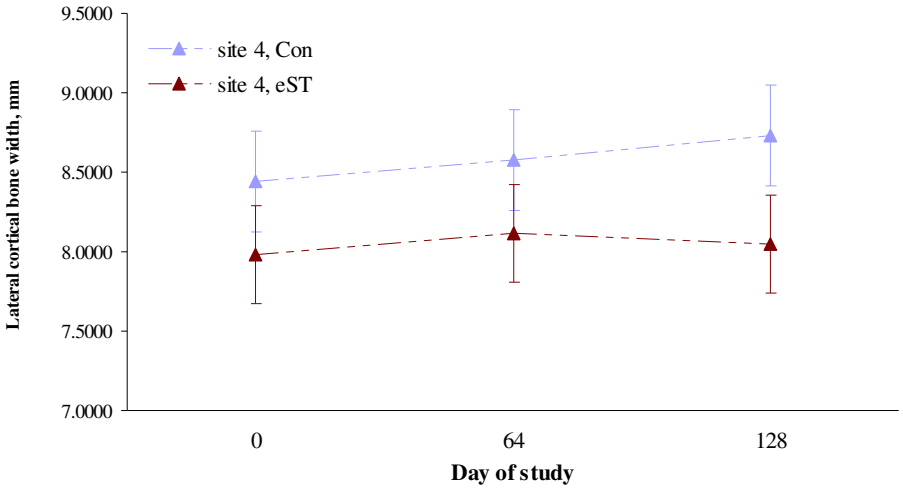


Figure 88. Lateral cortical bone width in mm, at site 4, vs day of study.

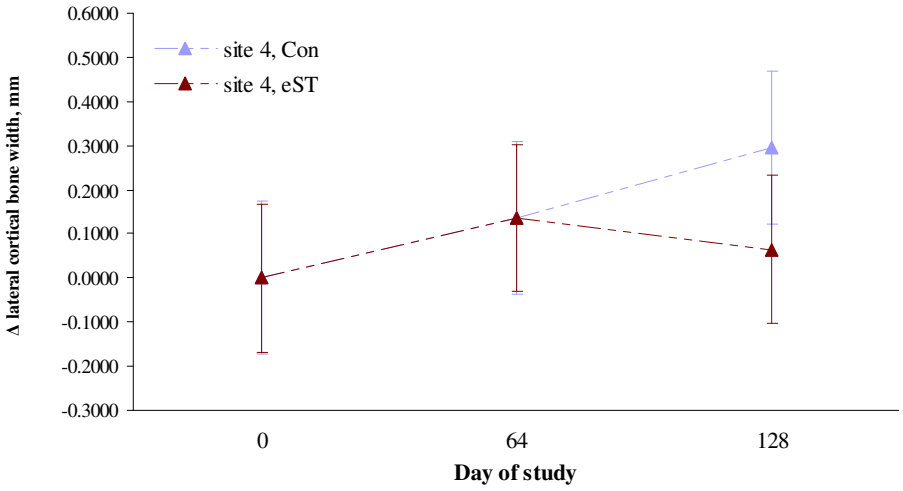


Figure 89. Change in lateral cortical bone width in mm, at site 4, vs day of study.

The data from the lateral cortical bone width at site 4 were normalized to day 0 and re-evaluated to more closely evaluate any changes that may have occurred between the

two treatment groups over the course of the project (Figure 89, Tables A-22 and B-88). There were no effects of day or treatment, nor where there any day*treatment interaction affects, on the changes in the width of the lateral cortical bone at site 4. There were no significant differences between the two treatment groups, but there was a pattern for the change in lateral cortical bone width to vary between the two treatment groups during the last half of the trial. From day 64 to day 128, the Con group of horses had an increase in the width of the lateral cortex, which is in direct contrast to the decrease in the width of the lateral cortex seen in the eST group of horses during this same time period.

Medial Cortical Bone Width Micrometer Readings

Site 1

There were significant day effects (P=.02), but no treatment effects nor any day*treatment interaction on the medial cortical bone width at site 1 (Figure 90, Tables A-23 and B-89). The width of the medial cortex at site 1 did not change from day 0 to day 64, but did increase significantly (P=.008) from day 64 to day 128. The Con group started the project with a non-significant greater width of the medial cortex than did the eST group, and this difference was still present on day 64. From day 64 to day 128, the Con group had no change in the medial cortical width at site 1, while in contrast, the eST group had a significant (P=.004) increase in the medial cortical width.

The data from the medial cortical bone width at site 1 were normalized to day 0 values and statistics were re-run (Figure 91, Tables A-24 and B-90). Day significantly (P=.02) effected the change in the width of the medial cortex at site 1, but treatment did not, nor was there a day*treatment interaction. There was no change in the medial bone width from day 0 to day 64, but a significant increase in the medial bone width occurred from day 64 to day 128 (P=.02). The two treatment groups had a trend to differ from each other by day 128 (P=.100) due to a greater increase in the width of the medial cortical bone from day 64 to day 128 in the eST group than in the Con group. On day 128 the change in the width of the medial cortex in the eST group was significantly greater than that on day 0 (P=.004) and day 64 (P=.004), while the Con group did not change significantly during the course of the project.

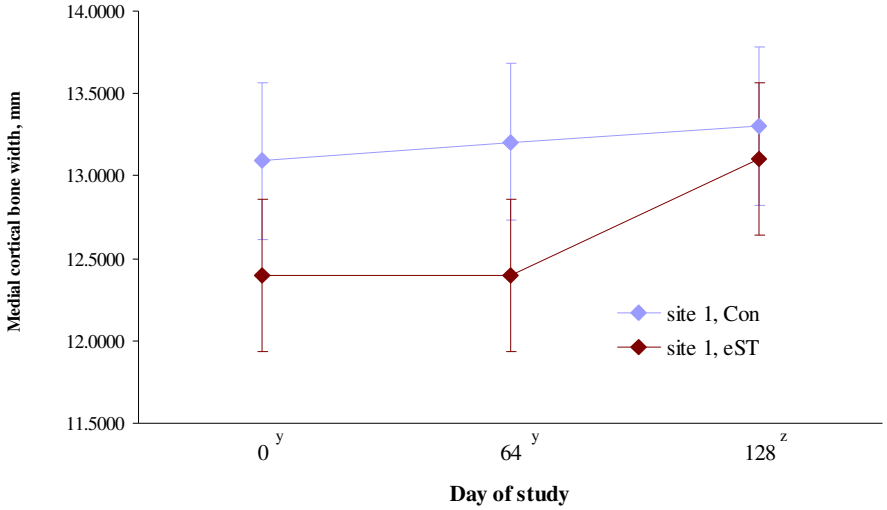


Figure 90. Medial cortical bone width in mm, at site 1, vs day of study.

[yz] Days not sharing the same superscript differ (P≤.05).

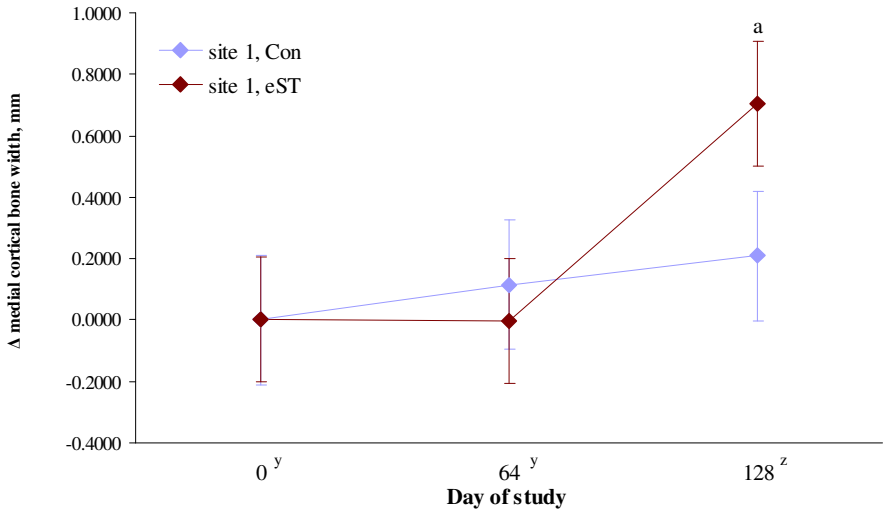


Figure 91. Change in medial cortical bone width in mm, at site 1, vs day of study.

[a] Trend for treatments to differ (P≤.10).

[yz] Days not sharing the same superscript differ (P≤.05).

Site 2

The medial cortical bone width at site 2 was significantly affected by day (P=.002), but was not affected by treatment or by any day*treatment interaction (Figure 92, Tables A-23 and B-91). The medial cortical bone width did not change from day 0 to day 64, then increased significantly (P=.008) from day 64 to day 128. The Con group of horses had a non-statistically greater width of the medial cortex at site 2 than did the eST group of horses that was present on day 0 and continued through the end of the trial on day 128. The Con group of horses had a non-statistical gain in the width of the medial cortex from day 0 to day 64, and from day 64 to day 128, with a significant increase in the width of the medial cortex from day 0 to day 128 (P=.05). The eST group of horses had a non-statistical gain in the width of the medial cortex from day 0 to day 64, and a signficant gain from day 64 to day 128 (P=.01).

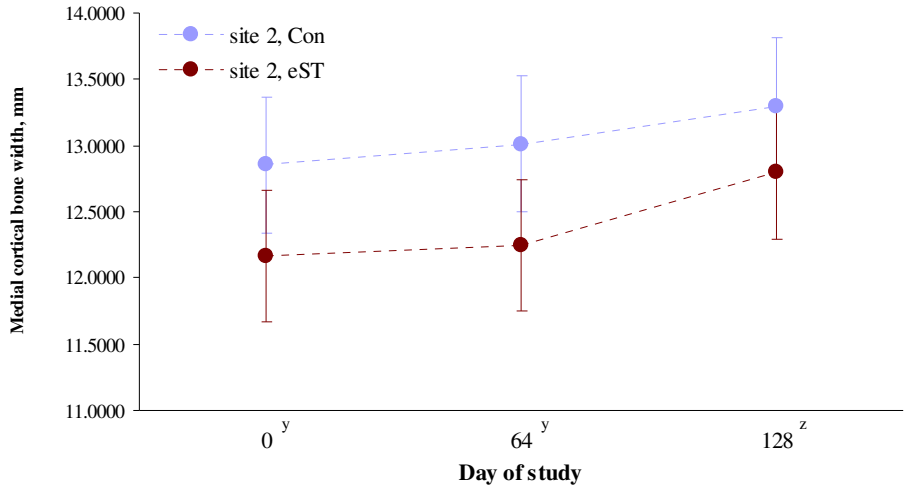


Figure 92. Medial cortical bone width in mm, at site 2, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.01).

Data from the medial cortical bone width at site 2 were normalized to day 0 values and re-evaluated to better examine any differences in the changes in the bone width over

the course of the experiment (Figure 93, Tables A-24 and B-92). Changes in the medial cortical bone width were significantly affected by day (P=.002), but not by treatment or by a day*treatment interaction. There was no significant change in the medial cortical bone width at site 2 from day 0 to day 64. There was a significant increase in the width of the medial cortex from day 64 to day 128 (P=.008). The eST group did have a greater gain in cortical bone width than the Con group from day 64 to day 128, but this difference was not significant.

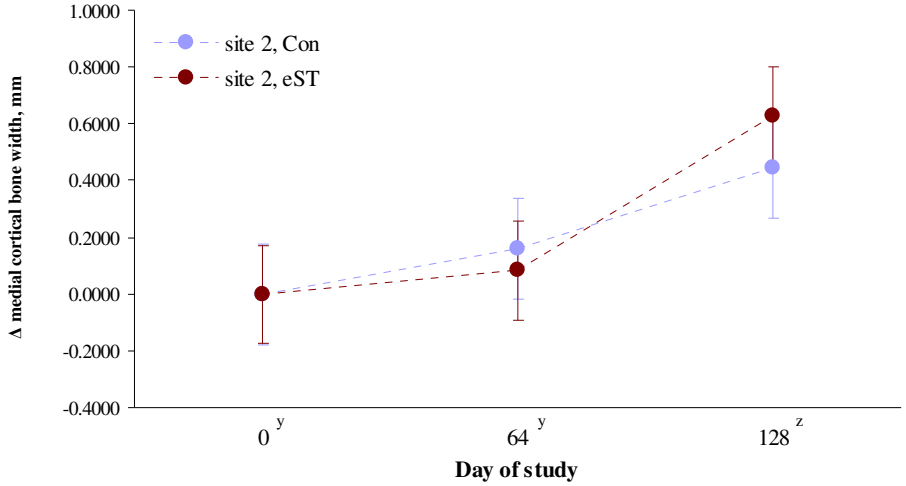


Figure 93. Change in medial cortical bone width in mm, at site 2, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.05).

Site 3

Medial cortical bone width at site 3 was not affected by treatment or by day*treatment interaction, but was significantly (P=.02) affected by day (Figure 94, Tables A-23 and B-93). There was not any statistically important change in the medial cortical bone thickness at site 3 from day 0 to day 64. A statistical gain in bone width in the medial cortex did occur between day 64 and day 128 (P=.04). The Con group had a non-

statistically greater width in the medial cortex than the eST group at the start of the project that continued to day 128.

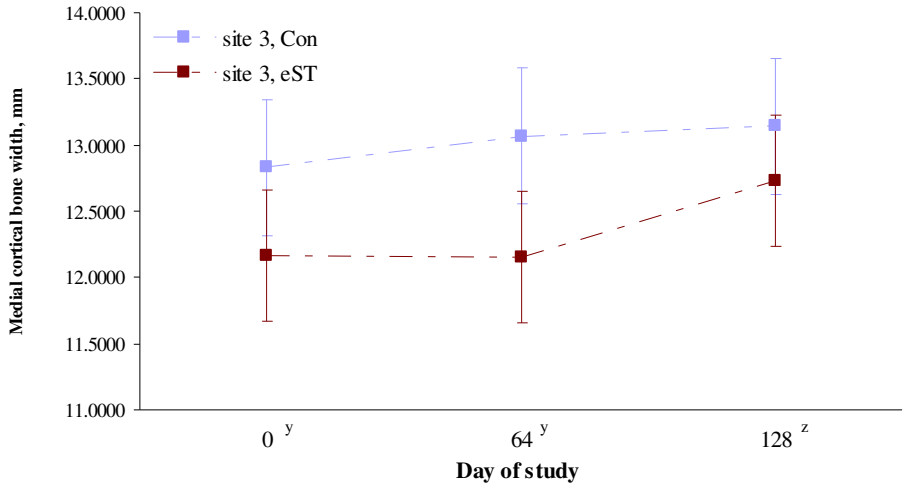


Figure 94. Medial cortical bone width in mm, at site 3, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.05).

To better evaluate the changes that occurred in the thickness of the medial cortex at site 3 over the course of the trial, these data were normalized to day 0 and re-evaluated (Figure 95, Tables A-24 and B-94).

Day effects were significant (P=.02), but there was no affect on the medial cortical bone width at site 3 due to treatment or to any day*treatment interaction. The medial cortical width did not change from day 0 to day 64, then increased significantly from day 64 to day 128 (P=.04). The pattern of change was different between the two treatment groups, though the two treatment groups did not differ significantly from each other on any given day. The Con group had a small, non-significant, increase in the medial cortical width from day 0 to day 64, and then no real change in the width from day 64 to day 128. The eST group, however, had a small, non-significant, decrease in the medial cortical width from day 0 to day 64, and then had a significant gain in the medial cortical width from day 64 to day 128 (P=.01).

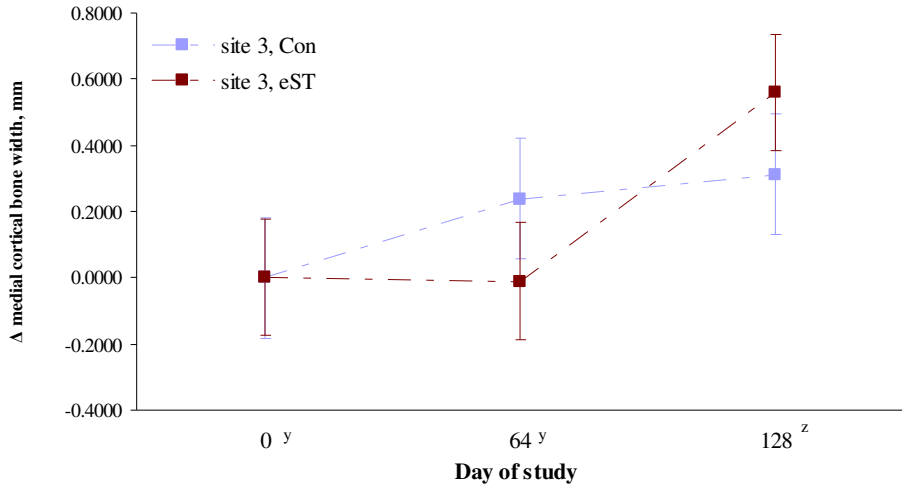


Figure 95. Change in medial cortical bone width in mm, at site 3, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.05).

Site 4

Medial cortical bone width at site 4 was not significantly affected by day, treatment, or by any day*treatment interaction (Figure 96, Tables A-23 and B-95). There was a trend for day 128 to have a greater medial cortical bone width than day 0 (P=.100). The two treatment groups did not differ significantly from each other on any given day, but there did appear to be a difference between the two groups. The Con group had no change in the width of the medial cortex from day 0 to day 128. The eST group had a significant increase in the width of the medial cortex from day 0 to day 128 (P=.01).

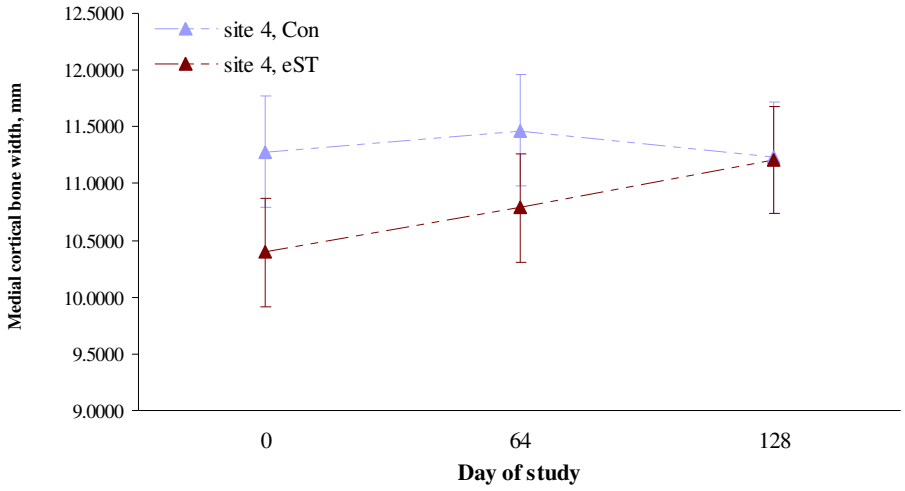


Figure 96. Medial cortical bone width in mm, at site 4, vs day of study.

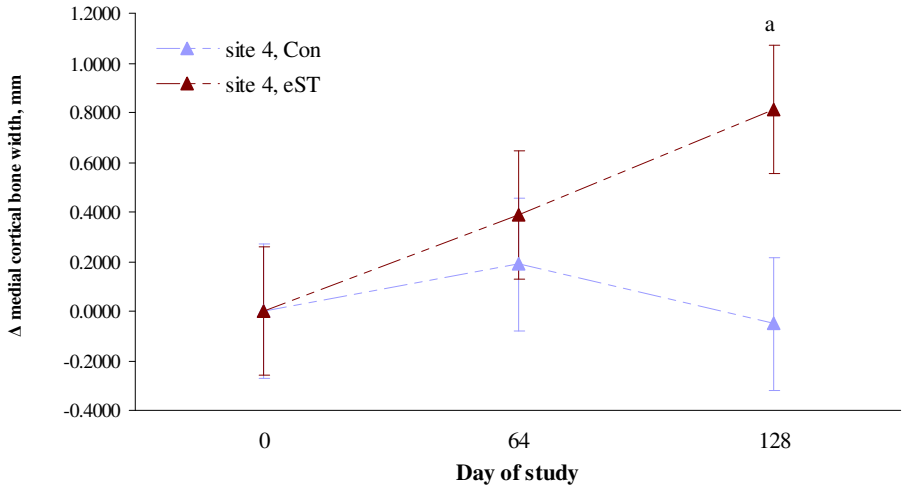


Figure 97. Change in medial cortical bone width in mm, at site 4, vs day of study.
[a] Treatments differ (P≤.05).

Medial cortical bone width data from site 4 were normalized to day 0 and statistics were run on the resulting data (Figure 97, Tables A-24 and B-96). There were no

significant effects on the change in medial cortical bone width at site 4 due to treatment, day, or to day*treatment interaction. There were no significant overall changes in the medial cortical bone width, but there was a trend for day 128 to have a greater overall medial cortical bone width at site 4 than day 0 (P=.100). The Con group of horses had a non-significant gain in the thickness of the medial cortex from day 0 to day 64 and a non-significant loss in the thickness of the medial cortex from day 64 to day 128, resulting in day 128 having a less thick medial cortex than day 0, though the two were not statistically different. The eST group of horses had a continual gain in the thickness of the medial cortical bone at site 4, resulting in day 128 values that were significantly greater than those on day 0 (P=.01). The eST group had a significantly greater gain in medial cortical bone width at site 4 on day 128 than did the Con group (P=.02).

Palmar Cortical Bone Width Micrometer Readings

Site 1

There were no day effects, treatment effects, or any day*treatment interaction that significantly affected the palmar cortical bone width at site 1 (Figure 98, Tables A-25 and B-97). There were not any overall changes nor were there any significant differences between the two treatment groups in the palmar cortical bone width at site 1.

Data from the palmar cortical bone width at site 1 were normalized to day 0 values and statistics were run on the resulting data to better observe any significant changes in the bone thickness at that site over the course of the experiment (Figure 99, Tables A-26 and B-98). There were still no significant treatment, day, or day*treatment effects on the palmar cortical bone width at site 1. There were no significant differences between the two treatment groups, though there was a pattern for the Con group to have a decrease in the thickness of the palmar cortex at site 1 over the course of the trial, while the eST group gained in palmar cortical thickness during the same time period.

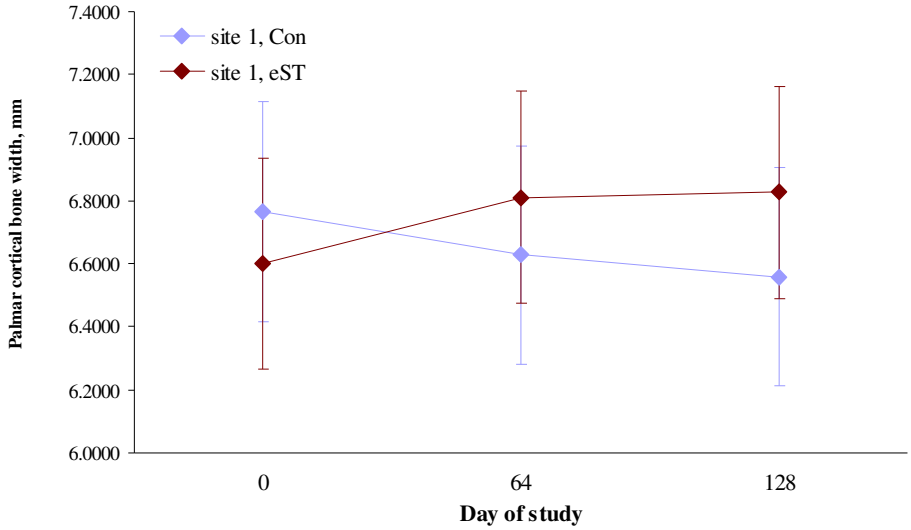


Figure 98. Palmar cortical bone width in mm, at site 1, vs day of study.

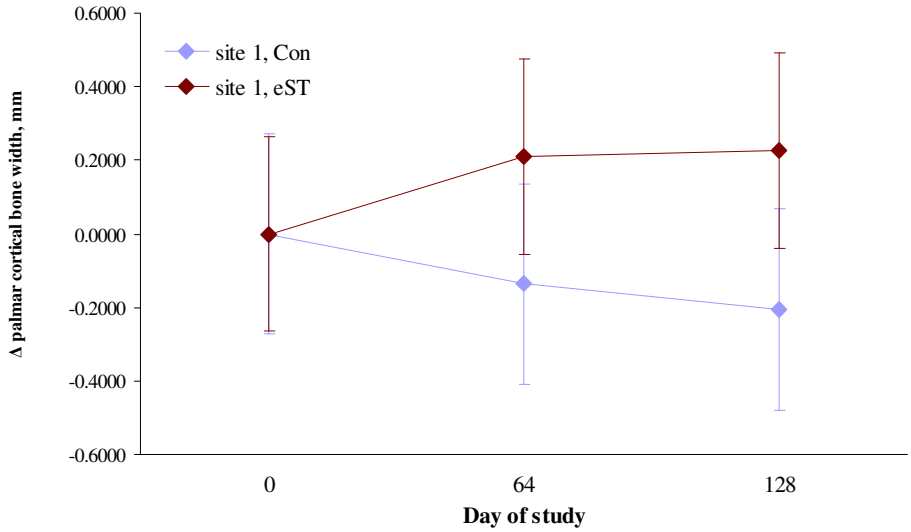


Figure 99. Change in palmar cortical bone width in mm, at site 1, vs day of study.

Site 2

There were no significant affects on the palmar cortical bone width at site 2 due to day, treatment, or to any day*treatment interaction (Figure 100, Tables A-25 and B-99). The two treatments did not differ from each other statistically at this site.

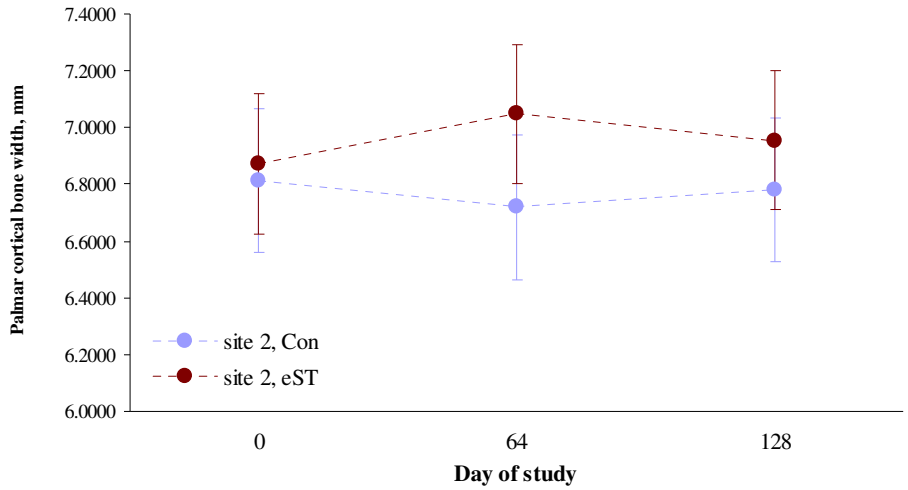


Figure 100. Palmar cortical bone width in mm, at site 2, vs day of study.

The data from the palmar cortical bone width at site 2 were normalized to day 0 values and the resulting data were analyzed statistically to determine if any treatment or day differences could be elicited in that manner (Figure 101, Tables A-26 and B-100). Once again, no treatment, day, or day*treatment effects were seen in the width of the palmar cortex at site 2. No significant differences occurred between the two treatment groups.

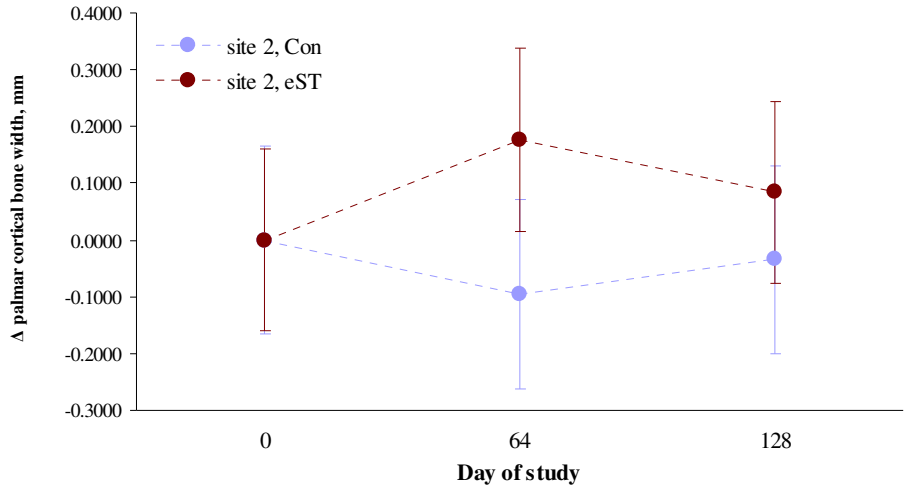


Figure 101. Change in palmar cortical bone width in mm, at site 2, vs day of study.

Site 3

Palmar cortical bone width at site 3 was not significnatly affected by treatment, day, or day*treatment interaction (Figure 102, Tables A-25 and B-101). The thickness of the palmar cortical bone at site 3 did not change significantly during the experiment. The eST group had a non-significantly thinner palmar cortical wall on day 0 than did the Con group. This had reversed by day 64, but all the differences between the two groups were well within normal deviations, and were not statistically important.

Data from the palmar cortical bone width at site 3 were normalized to day 0 values and statistics were run on the resulting data (Figure 103, Tables A-26 and B-102). Similar to the raw data, the normalized data showed that there were no significant affects on any changes in the palmar cortical bone width at site 3 due to treatment, day, or day*treatment interaction. The eST group did have a non-significant gain in the width of the palmar cortex from day 0 to day 64 while the Con group had a non-significant decrease in the width of the palmar cortex from day 0 to day 64, but the two treatment groups were not significantly different from each other on any of the days.

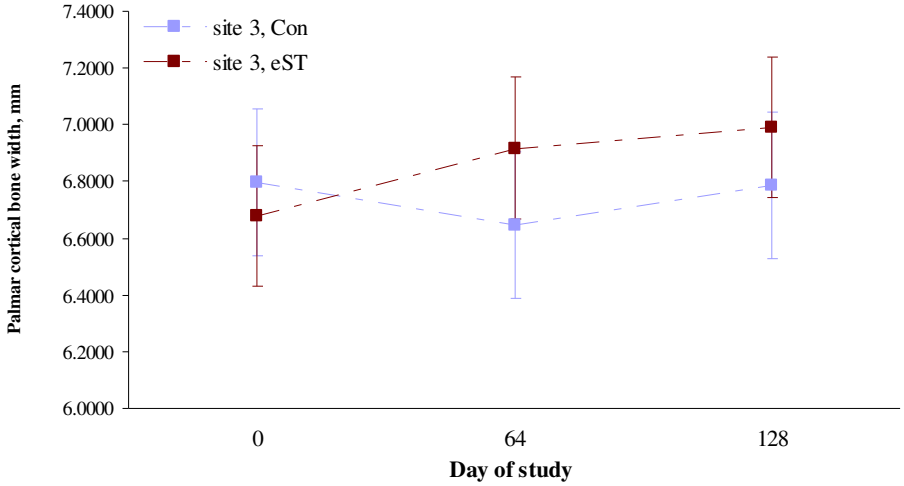


Figure 102. Palmar cortical bone width in mm, at site 3, vs day of study.

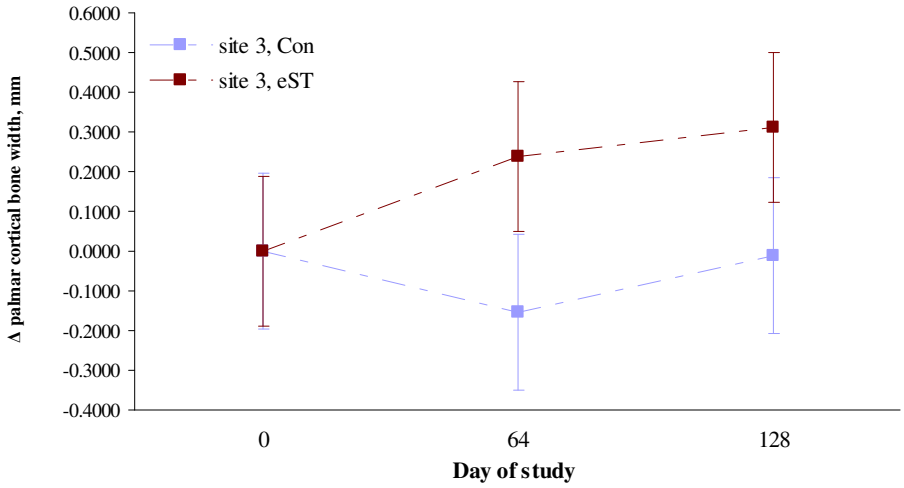


Figure 103. Change in palmar cortical bone width in mm, at site 3, vs day of study.

Site 4

The palmar cortical bone width at site 4 was significantly affected by day, though not by treatment or by any day*treatment interaction (Figure 104, Tables A-25 and B-103). There was a gradual, continual gain in the width of the palmar cortical bone through the trial. By day 128, the width of the palmar cortical bone at site 4 was significantly greater than it had been on day 0 (P<.0001) or on day 64 (P=.005). Within the treatment groups, the Con group had no change in the thickness of the palmar cortex at site 4 from day 0 to day 64, then a trend for an increase in the thickness of the cortex from day 64 to day 128 (P=.07), with the cortex on day 128 being significantly wider than it had been on day 0 (P=.03). The eST group had a more rapid increase in palmar cortical thickness, with a trend for significant increase from day 0 to day 64 (P=.07), a significant increase from day 64 to day 128 (P=.03), and day 128 values being greater than those seen on day 0 (P=.0002). The two treatment groups did not significantly differ from each other.

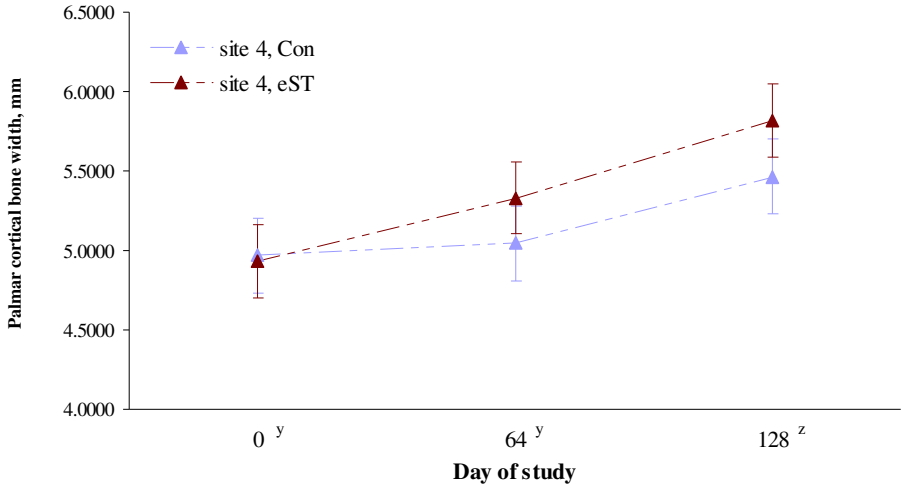


Figure 104. Palmar cortical bone width in mm, at site 4, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.01).

Palmar cortical bone width at site 4 data were normalized to remove day 0 values and were then re-evaluated (Figure 105, Tables A-26 and B-104). Day effects were significant (P=.0002), but treatment effects and day*treatment interaction were not statistically important. There was a non-significant increase in the palmar cortical bone width at site 4 from day 0 to day 64 that reached significance from day 64 to day 128 (P=.005). The width of the palmar cortex at site 4 was significantly greater on day 128 than on day 0 (P<.0001). The two treatment groups did not statistically differ from each other. The Con group of horses had no change in palmar cortical thickness from day 0 to day 64, a trend for an increase from day 64 to day 128 (P=.07), and a significant gain in cortical thickness from day 0 to day 128 (P=.03). The eST group of horses had a more consistent increase in the palmar cortical thickness at site 4 that lasted the full course of the project, with the increase from day 0 to day 64 having a trend toward significance (P=.07), a significant increase from day 64 to day 128 (P=.03), and a significant gain in cortical width from day 0 to day 128 (P=.0002).

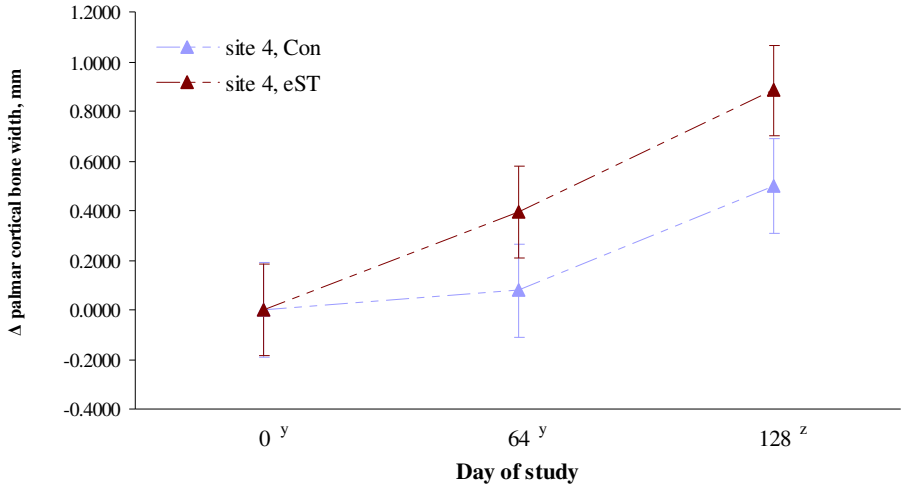


Figure 105. Change in palmar cortical bone width in mm, at site 4, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.01).

Dorsal to Palmar Medullary Cavity Width Micrometer Readings

Site 1

The width of the medullary cavity in the dorsal to palmar direction at site 1 was significantly affected by day (P=.006), but not by treatment or by a day*treatment interaction (Figure 106, Tables A-27 and B-105). The width of the medullary cavity decreased through the experiment. The decrease that occurred from day 0 to day 64 was not significant, the decrease that occurred from day 64 to day 128 trended toward being significant (P=.08), and the overall decrease from day 0 to day 128 was significant (P=.001). The decrease in medullary cavity width in the dorsal to palmar direction at site 1 was not significant in the Con group of horses. The eST group of horses, in contrast, had a decrease in the medullary cavity width in the dorsal to palmar direction at site 1 that had a trend to be significant from day 0 to day 64 (P=.09), and was significant from day 64 to day 128 (P=.03) and from day 0 to day 128 (P=.0003). The differences between the two treatment groups were not significant.

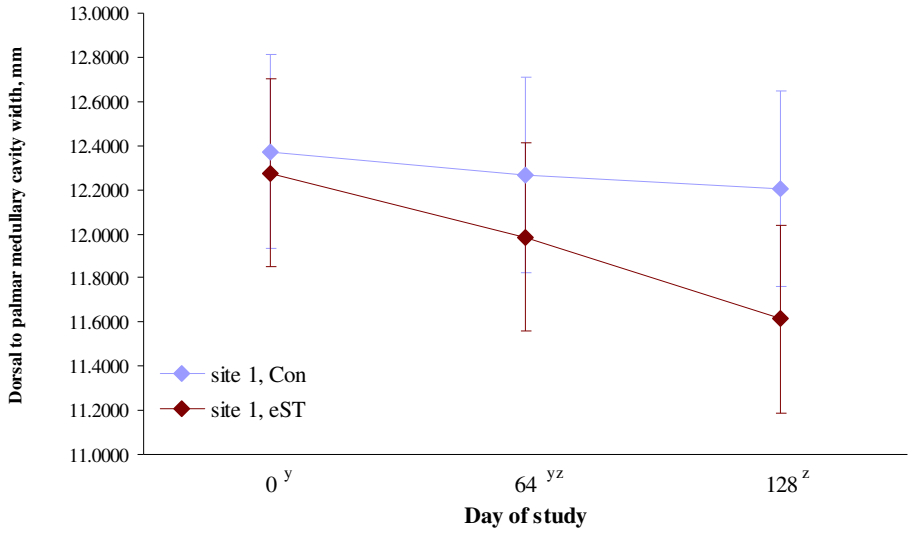


Figure 106. Dorsal to palmar medullary cavity width in mm, at site 1, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.01).

Data from the width of the medullary cavity in the dorsal to palmar direction at site 1 were normalized to remove day 0 values and better evaluate changes that occurred over the course of the trial (Figure 107, Tables A-28 and B-106). Day effects were significant (P=.005), but treatment and day*treatment interaction did not affect the meduallary cavity width at this site. There was an overall pattern of decrease in the width of the medullar cavity at this site that was not significant from day 0 to day 64, had a trend for significance from day 64 to day 128 (P=.08), and was significant from day 0 to day 128 (P=.001). The Con group of horses had a decrease in medullary cavity width that never did reach significance. In contrast, the decrease in the medullary cavity width in the eST group of horses trended toward significance from day 0 to day 64 (P=.09), and was significant from day 64 to day 128 (P=.03) and from day 0 to day 128 (P=.0003). The eST group of horses had a significantly greater decrease in the width of the medullary cavity than did the Con group of horses on day 128 (P=.02).

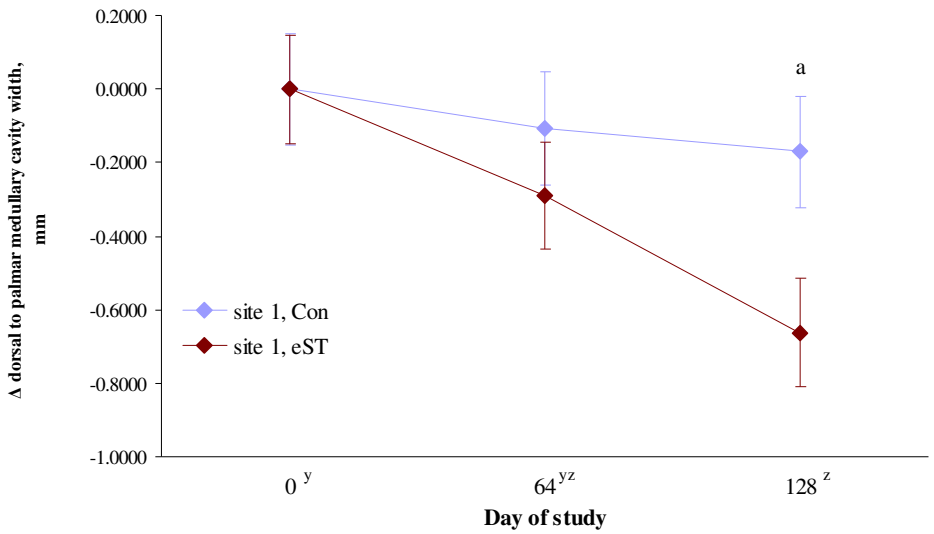


Figure 107. Change in dorsal to palmar medullary cavity width in mm, at site 1, vs day of study.

[a] Treatments differ (P≤.05).

[yz] Days not sharing the same superscript differ (P≤.05).

Site 2

There were significant day effects (P=.0005), a trend for an effect due to day*treatment interaction (P=.07), and no significant treatment effect on the width of the dorsal to palmar medullary cavity at site 2 (Figure 108, Tables A-27 and B-107). There was a decrease in the medullary cavity width that was significant from day 0 to day 128 (P<.0001), day 0 to day 64 (P=.04, and day 64 to day 128 (P=.04). The decrease in medullary width at site 2 in the Con group did not reach significance. The decrease in medullary width at site 2 in the eST group was significant from day 0 to day 64 (P=.005), day 0 to day 128 (P<.0001), and trended toward significance from day 64 to day 128 (P=.100). The two treatment groups did not significantly differ from each other.

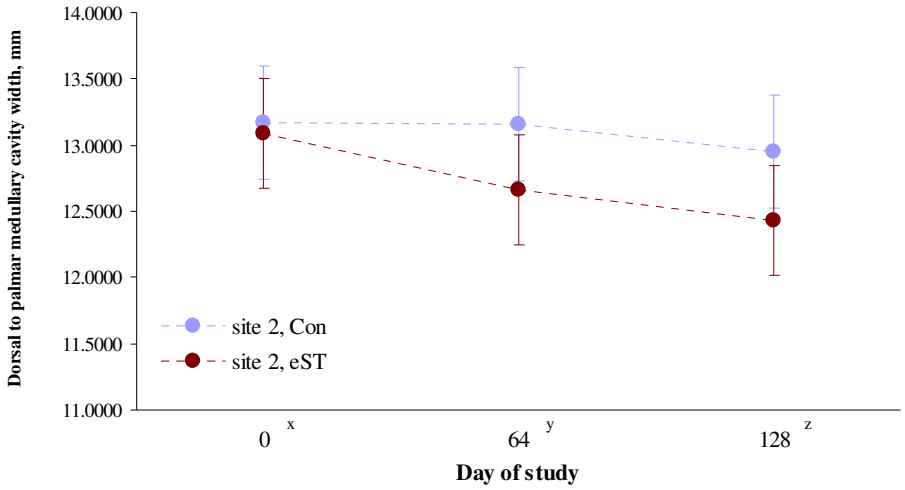


Figure 108. Dorsal to palmar medullary cavity width in mm, at site 2, vs day of study.
^{xyz} Days not sharing the same superscript differ (P≤.05).

The data from the dorsal to palmar medullary cavity width at site 2 were normalized to remove day 0 values and to more closely examine the changes in the medullary width that occurred over the course of the research project (Figure 109, Tables A-28 and B-108). Treatment effects (P=.03) and day effects (P=.0005) were significant, and there

was a trend (P=.07) for day*treatment interaction to effect the change in the dorsal to palmar medial cavity width at site 2. There was a significant decrease in the medullary cavity width from day 0 to day 64 (P=.04), day 64 to day 128 (P=.04), and day 0 to day 128 (P<.0001). The Con group had no significant change in the width of the medullary cavity during the experiment. The eST group, in contrast, had a trend for a decrease from day 64 to day 128 (P=.100), and a significant decrease in the width of the medullar cavity at site 2 from day 0 to day 64 (P=.005), day 0 to day 128 (P<.0001). The two treatment groups significantly differed from each other on day 64 (P=.02) and day 128 (P=.01) due to the greater decrease in the dorsal to palmar medullary cavity width at site 2 in the eST group as compared to the Con group.

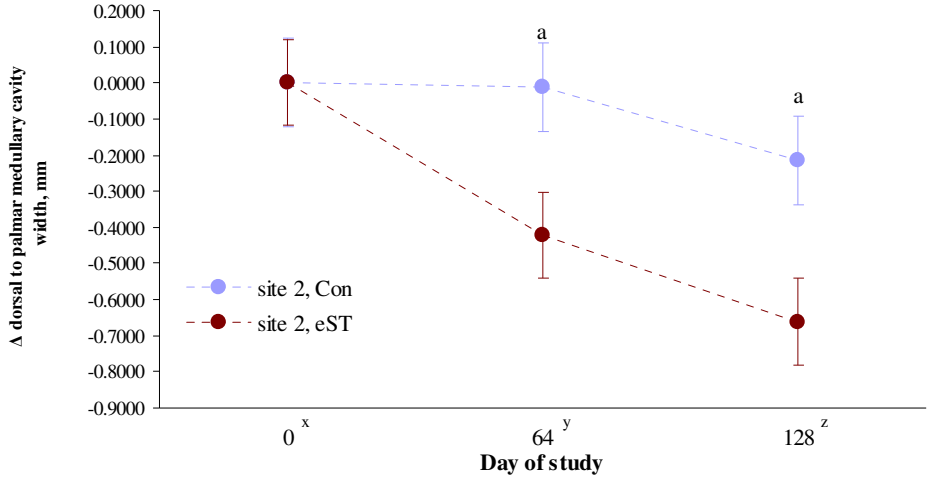


Figure 109. Change in dorsal to palmar medullary cavity width in mm, at site 2, vs day of study.

[a] Treatments differ (P≤.05).

[xyz] Days not sharing the same superscript differ (P≤.05).

Site 3

The dorsal to palmar medullary cavity width at site 3 was not significantly affected by day, treatment, or by day*treatment interaction (Figure 110, Tables A-27 and B-109). There was a decrease in the medullary cavity width at site 3 from day 0 to day 128 that

trended toward significance (P=.100). The Con group had no change in the dorsal to palmar diameter of the medullary cavity at site 3 during this experiment. The eST group had a significant decrease in the dorsal to palmar medullary cavity width from day 0 to day 128 (P=.01). The differences between the two treatment groups were not significant.

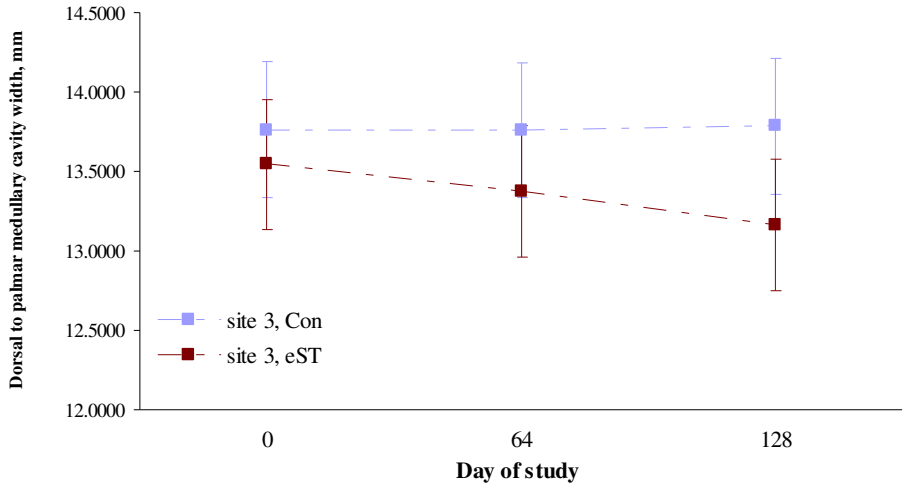


Figure 110. Dorsal to palmar medullary cavity width in mm, at site 3, vs day of study.

Data from the dorsal to palmar medullary cavity width at site 3 were normalized to day 0 values and the resulting normalized data were statistically analyzed to better evaluate any changes in the medullary width over the course of the trial (Figure 111, Tables A-28 and B-110). There were still no significant affects on the medullary cavity width due to day, treatment, or day*treatment interaction. A decrease in the medullary cavity width was not significant from day 0 to day 64 or day 64 to day 128, but did have a trend toward significance from day 0 to day 128 (P=.100). No significant changes occurred in the dorsal to palmar medullary cavity width in the Con group during the trial. A decrease in the medullary cavity width in the eST group was not significant from day 0 to day 64, or from day 64 to day 128, but was significant from day 0 to day 128 (P=.01)

and resulted in a significant difference between the two treatment groups on day 128 (P=.02).

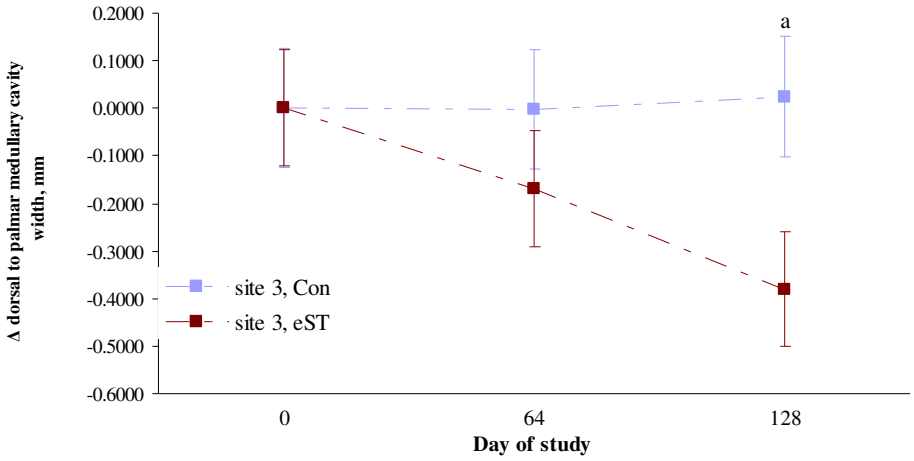


Figure 111. Change in dorsal to palmar medullary cavity width in mm, at site 3, vs day of study.
[a] Treatments differ (P≤.05).

Site 4

There were no significant treatment, day, or day*treatment effects on the dorsal to palmar medullary cavity width at site 4 (Figure 112, Tables A-27 and B-111). A minor decrease in the diameter of the medullary cavity did not reach significant levels. The Con group started the trial on day 0 with a non-statistically larger medullary cavity width at site 4 than did the eST group. There was no significant change in the medullary cavity width in the Con group during the project. The decrease in medullary cavity width in the eST group had a trend toward significance by day 128 (P=.07).

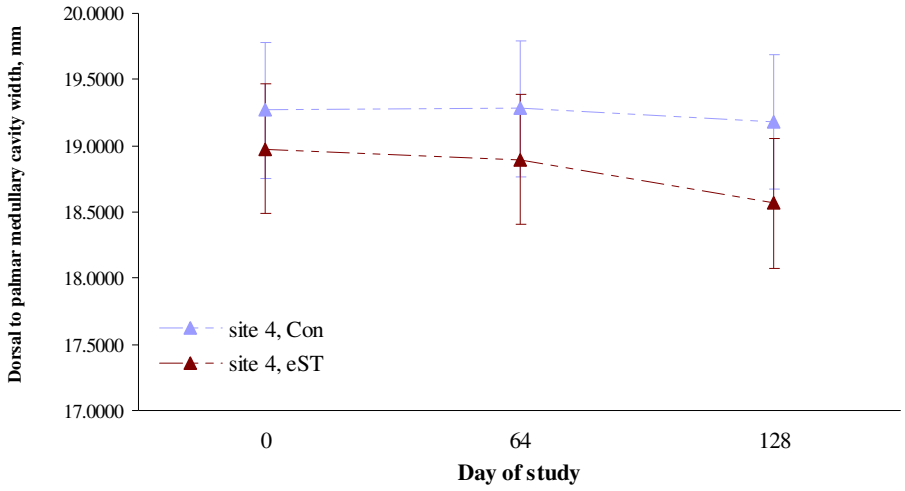


Figure 112. Dorsal to palmar medullary cavity width in mm, at site 4, vs day of study.

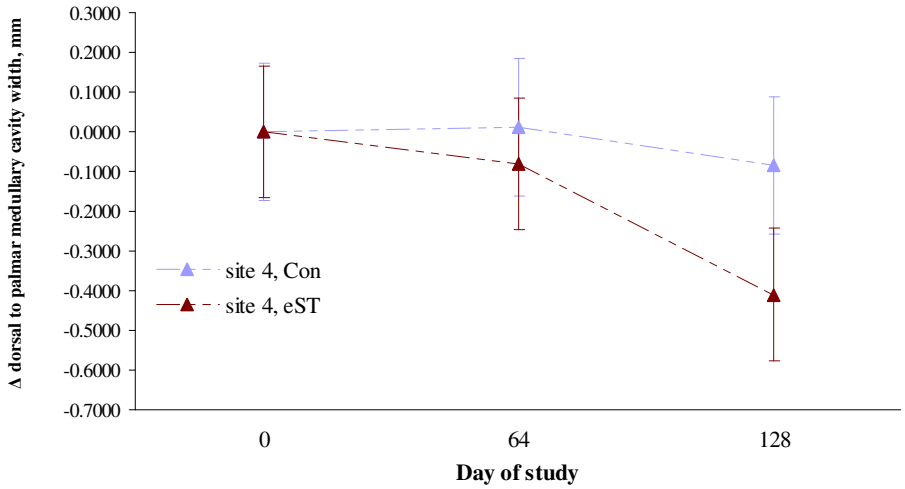


Figure 113. Change in dorsal to palmar medullary cavity width in mm, at site 4, vs day of study.

The data from the dorsal to palmar medullary cavity width at site 4 were normalized to day 0 values and statistics were re-run (Figure 113, Tables A-28 and B-112). Similar to the raw data at this site (Figure 112), no significant changes in the medullary cavity width resulted from treatment, day, or any day*treatment interaction. No significant overall changes in the medullary cavity width occurred. No significant differences were seen between the two treatment groups. The eST group did have a trend (P=.07) for the decrease in the medullary cavity diameter from day 0 to day 128 to be important, and this was not seen in the Con group.

Dorsal to Palmar Bone Diameter Micrometer Readings

Site 1

There was a significant day effect (P=.002), but not a treatment effect or a day*treatment interaction that affected the dorsal to palmar bone diameter at site 1 (Figure 114, Tables A-29 and B-113). A non-significant gain in the diameter of the bone at site 1 from day 0 to day 64 increased to be a significant gain from day 64 to day 128 (P=.006). The diameter of the bone in the dorsal to palmar direction was significantly greater on day 128 than on day 0 (P=.0006). Though there was an increase in the diameter of the bone from day 0 to day 128 in the Con group, this increase was not significant. The increase in the eST group was significant, with the total width of the bone at site 1 on day 128 statistically different from day 64 (P=.003) and day 0 (P=.0003). The Con group had greater bone diameter than the eST group on day 0 and day 64, and less bone diameter than the eST group on day 128, but these differences were not great enough to be of statistical importance.

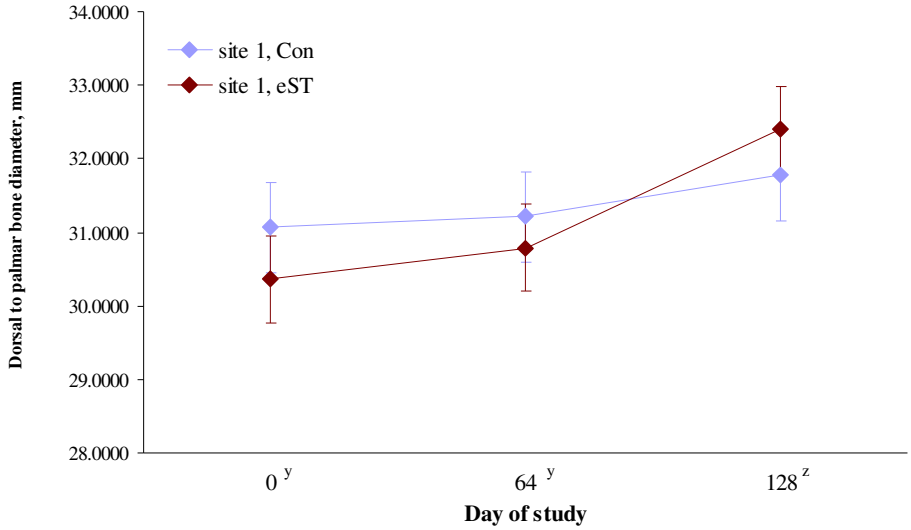


Figure 114. Dorsal to palmar bone diameter in mm, at site 1, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.01).

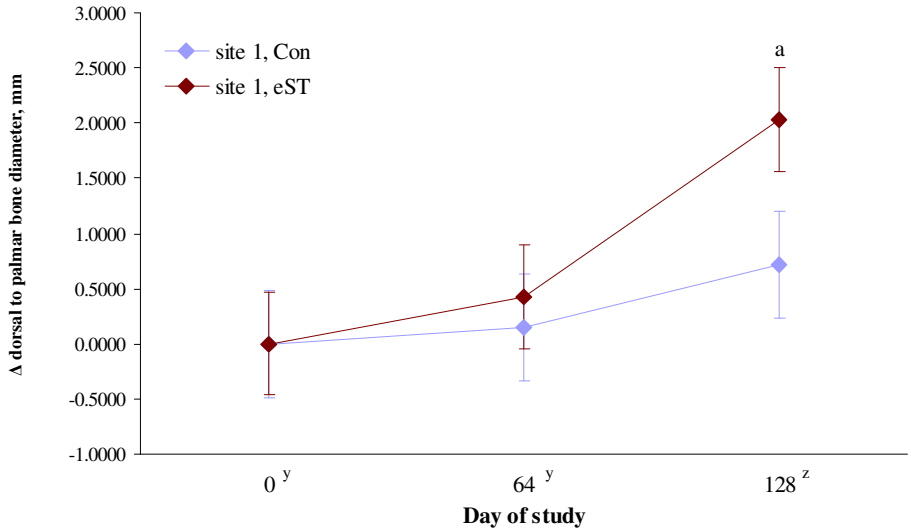


Figure 115. Change in dorsal to palmar bone diameter in mm, at site 1, vs day of study.
[a] Treatments differ (P≤.05).
[yz] Days not sharing the same superscript differ (P≤.05).

Treatment did not affect the normalized dorsal to palmar bone diameter at site 1, neither did any day*treatment interaction, however day effects were significant (Figure 115, Tables A-30 and B-114). A gradual increase in the bone width from day 0 to day 64 became more pronounced from day 64 to day 128 and resulted in day 128 values being significantly greater than those on day 0 (P=.0006) or on day 64 (P=.006). The Con group of horses had a non-significant increase in the diameter of the bone in the dorsal to palmar direction from day 0 to day 128. The eST group of horses had a steeper increase in the diameter of the bone that was significant from day 64 to day 128 (P=.003), and caused the day 128 values to be statistically greater than the values on day 0 (P=.0003). Additionally, the greater gain in bone diameter of the eST group of horses as compared to the Con group of horses was significant by day 128 (P=.05).

Site 2

Day effects were significant (P=.0001), but neither treatment nor day*treatment interaction significantly affected the dorsal to palmar bone diameter at site 2 (Figure 116, Tables A-29 and B-115). The width of the bone did not change significantly from day 0 to day 64 but did increase significantly after day 64, causing the bone on day 128 to have a greater diameter than on day 0 (P<.0001) or day 64 (P=.001). The Con group started the trial with a non-statistically wider bone at site 2 than did the eST group, but this was reversed by day 128 due to a greater increase in the width of the bone from day 64 to day 128 in the eST group than in the Con group. The increase in bone diameter from day 0 to day 128 was significant in the eST group (P<.0001), while the Con group only had a trend for significance (P=.07). The differences between the two treatment groups were not great enough to be significant.

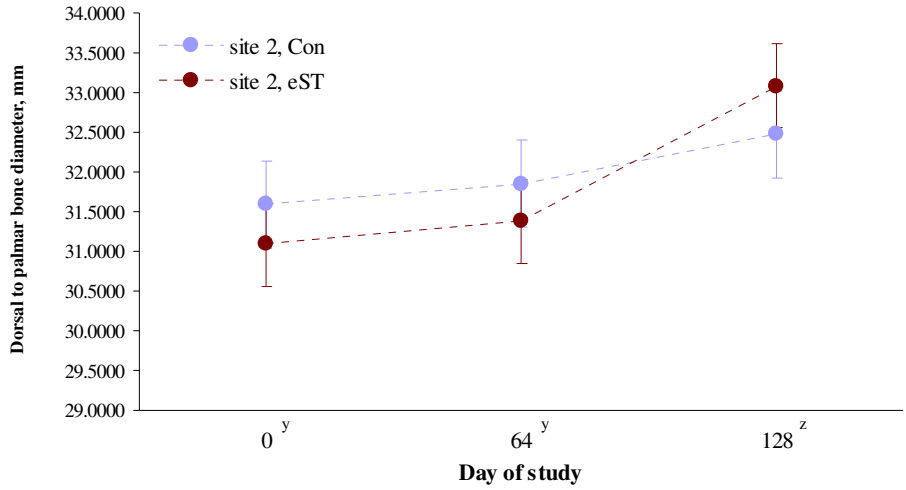


Figure 116. Dorsal to palmar bone diameter in mm, at site 2, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.01).

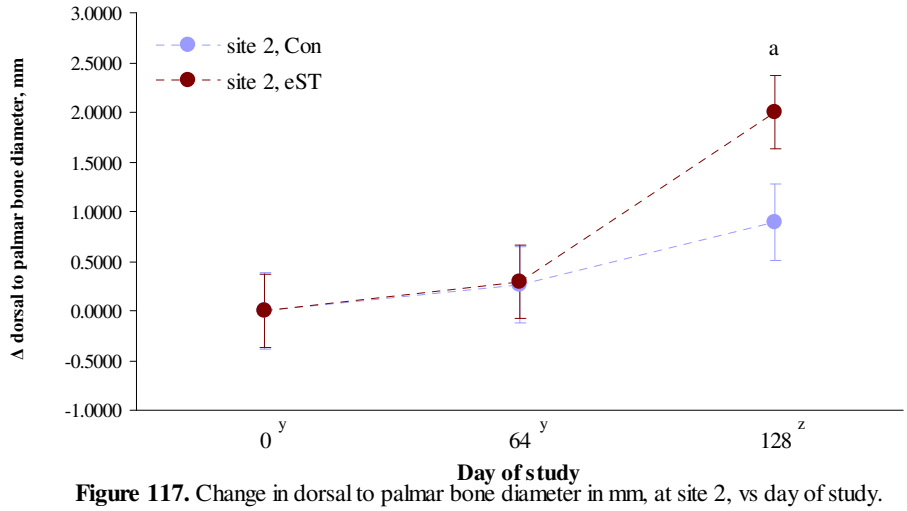


Figure 117. Change in dorsal to palmar bone diameter in mm, at site 2, vs day of study.
[a] Treatments differ (P≤.05).
[yz] Days not sharing the same superscript differ (P≤.05).

To better compare changes between the two treatment groups over the duration of the research trial, the data from the dorsal to palmar bone diameter at site 2 were normalized

by removing day 0 values and the resulting data were subjected to statistical analyses (Figure 117, Tables A-30 and B-116). Day effects were significant (P=.0001), but treatment effects and day*treatment interactions did not significantly affect the width of the bone at site 2. The increase in bone diameter at site 2 was significant during the last half of the experiment, consequentially the width of the bone on day 128 was significantly greater than on day 0 (P<.0001) or day 64 (P=.001). The two treatment groups did not differ from each other during the first half of the trial. During the second half of the trial, the increase in bone width in the Con group was not great enough to be significant while the increase in bone width in the eST group was significant (P=.0006). At the end of this project on day 128, the eST group had a significantly greater increase in the dorsal to palmar bone diameter at site 2 than did the Con group (P=.04).

Site 3

There were day effects (P<.0001) but no significant treatment or day*treatment effects on the dorsal to palmar bone diameter at site 3 (Figure 118, Tables A-29 and B-117). The increase in the bone diameter from day 0 to day 64 was not significant. The increase in bone diameter from day 64 to 128 was great enough that day 128 values were significantly larger than day 0 (P<.0001) or day 64 (P<.0001). In the Con group, the bone width on day 128 was significantly greater than on day 0 (P=.01) or on day 64 (P=.04). In the eST group, the bone width on day 128 was also significantly greater than on day 0 (P<.0001) or on day 64 (P<.0001). There were not significant differences between the two treatment groups on any given day.

The data from the bone diameter in the dorsal to palmar direction at site 3 were normalized to remove day 0 values and re-examined statistically to more closely evaluate changes that occurred during this experiment (Figure 119, Tables A-30 and B-118). There were significant day effects (P<.0001), but no effects due to treatment or to a day*treatment interaction. No significant increase in bone diameter at site 3 occurred from day 0 to day 64. From day 64 to day 128 there was a significant increase in total bone width at site 3 (P<.0001). The same pattern occurred in both treatment groups, with no real change in the bone width from day 0 to day 64, and a significant increase in

bone width from day 64 to day 128 in both the Con group (P=.04) and the eST group (P<.0001). The amount of increase in diameter of the bone at site 3 was greater in the eST group than the Con group, consequently the two groups differed from each other in the amount of gain on day 128 (P=.03).

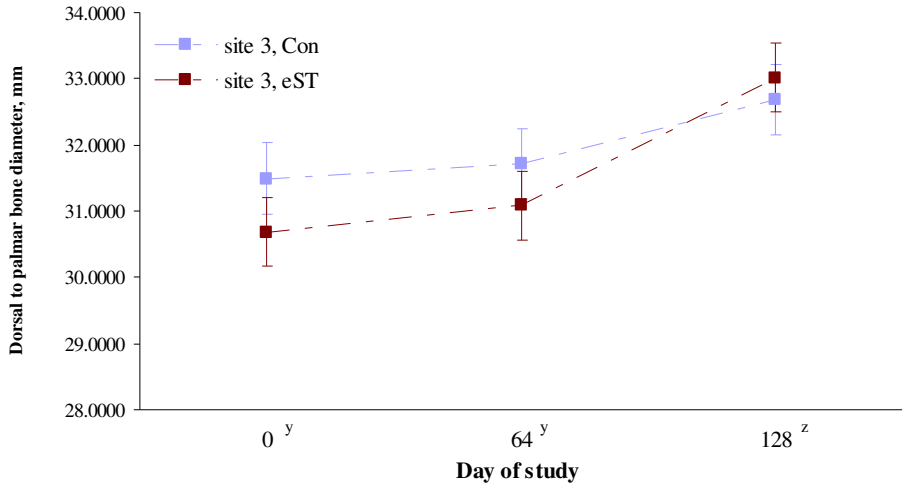


Figure 118. Dorsal to palmar bone diameter in mm, at site 3, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.01).

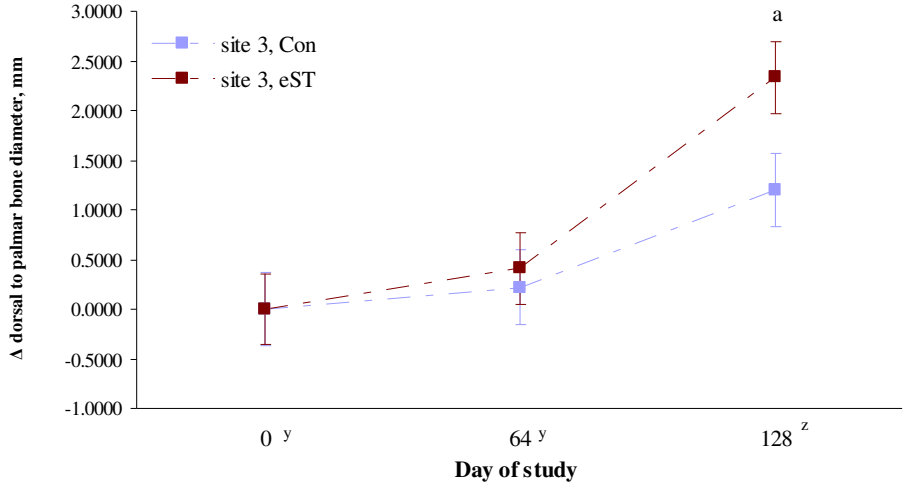


Figure 119. Change in dorsal to palmar bone diameter in mm, at site 3, vs day of study.
[a] Treatments differ (P≤.05).
[yz] Days not sharing the same superscript differ (P≤.05).

Site 4

There were no treatment effects, nor any day*treatment interaction that affected the bone width in the dorsal to palmar direction at site 4, but day effects were significant (P<.0001) (Figure 120, Tables A-29 and B-119). There was a trend for an increase in the bone width from day 0 to day 64 (P=.07) that became significant from day 64 to day 128 (P<.0001). A non-significant difference between the two groups on day 0 had disappeared by day 128. The Con group day 0 values were not significantly different from day 64, but were less than day 128 values (P<.0001), and day 64 values were significantly less than day 128 (P=.0006). The eST group day 0 values had a trend to be less than day 64 (P=.100) and were less than day 128 (P<.0001), and day 64 values were significantly less than day 128 (P<.0001).

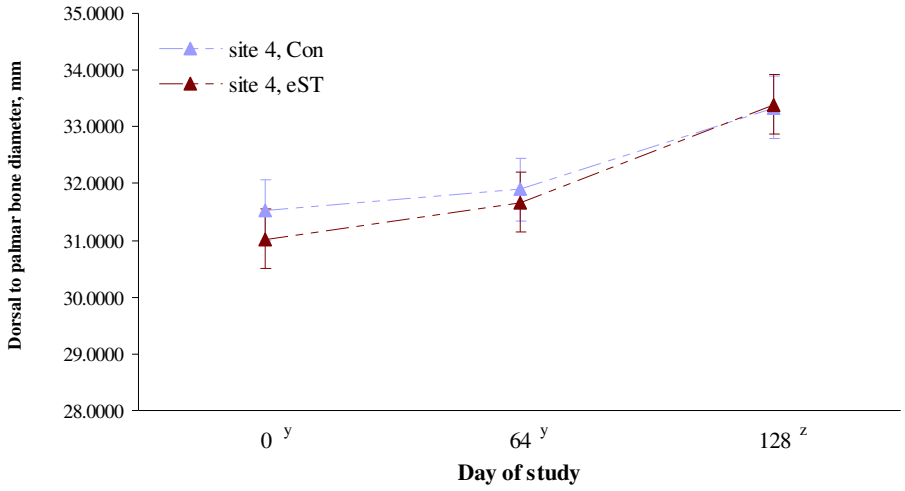


Figure 120. Dorsal to palmar bone diameter in mm, at site 4, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.05).

Data from the dorsal to palmar bone diameter at site 4 were normalized to day 0 values and statistics were run on the resulting data to better examine changes in the bone width over the course of the experiment (Figure 121, Tables A-30 and B-120). Day effects were significant (P<.0001), with the gain in bone width from day 0 to day 64 trending toward significance (P=.07), and the gain from day 64 to day 128 (P<.0001) and the gain from day 0 to day 128 (P<.0001) both being significant. Treatment effects did not significantly affect the bone width at site 4, nor did any day*treatment interaction. The Con group had a non-significant increase in the bone diameter from day 0 to day 64, that became significant from day 64 to day 128 (P=.0006). The increase in the bone diameter in the eST group trended toward significance from day 0 to day 64 (P=.100) and was significant from day 64 to day 128 (P<.0001). On day 128 the gain in the diameter of the bone at site 4 was greater in the eST group than in the Con group, but this difference was not statistically important.

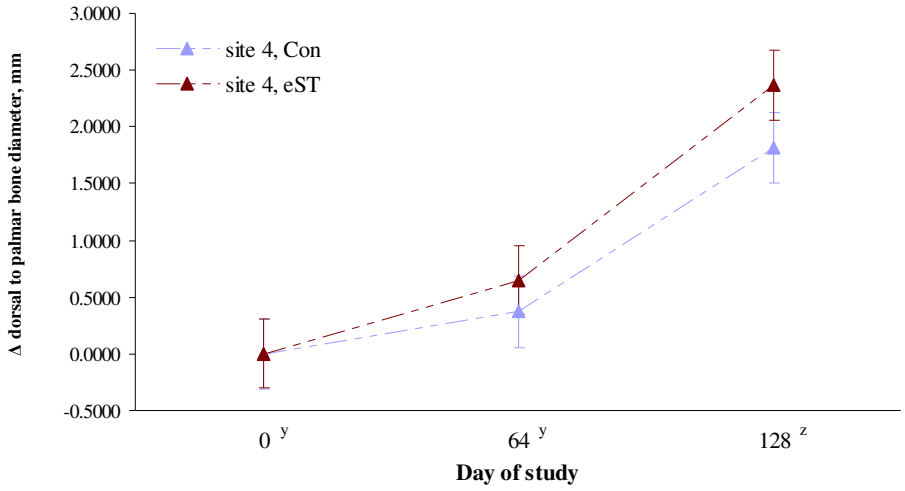


Figure 121. Change in dorsal to palmar bone diameter in mm, at site 4, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.05).

Lateral to Medial Medullary Cavity Width Micrometer Readings

Site 1

There were no significant effects on the lateral to medial medullary cavity width at site 1 due to day, treatment, or day*treatment interaction (Figure 122, Tables A-31 and B-121). No changes occurred in the width of the medullary cavity overall, or in either of the two treatment groups. The Con group did have a non-significantly greater width of the medullary cavity than did the eST group on day 0, and this pattern remained unchanged through day 128.

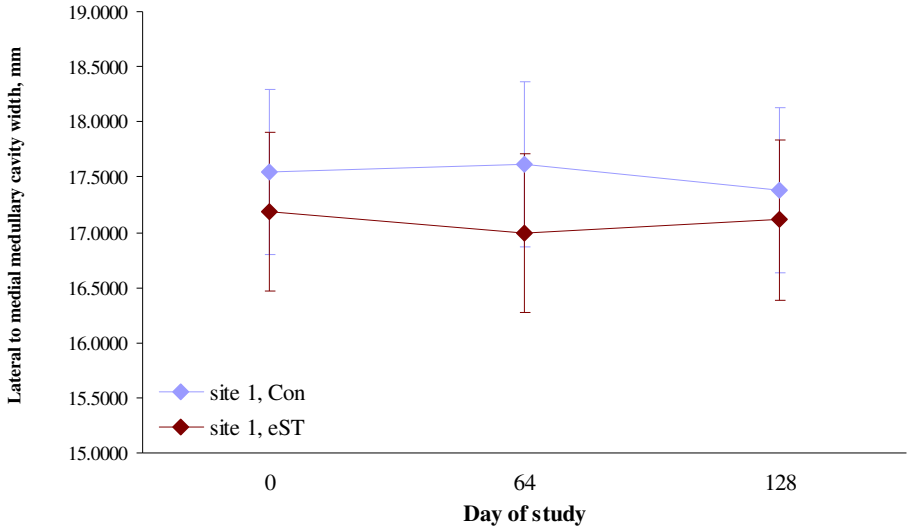


Figure 122. Lateral to medial medullary cavity width in mm, at site 1, vs day of study.

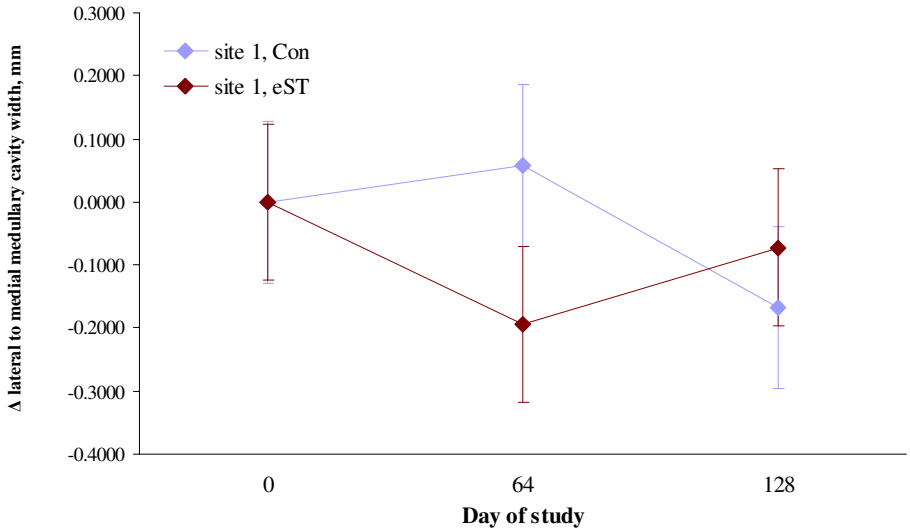


Figure 123. Change in lateral to medial medullary cavity width in mm, at site 1, vs day of study.

The data from lateral to medial medullary cavity width at site 1 were normalized to day 0 values and re-evaluated (Figure 123, Tables A-32 and B-122). There were no effects due to day, treatment, or any day*treatment interaction. The pattern of gain varied between the two treatment groups. The Con group enlarged the medullary cavity from day 0 to day 64, and then decreased the medullary cavity width to below day 0 values by day 128. The eST group had a decrease in the medullary cavity width from day 0 to day 64 followed by an increase in the width from day 64 to day 128. However, none of the changes, in either treatment group were significant, nor did the two treatment groups significantly differ from each other at any point during this trial in the diameter of the lateral to medial medullary cavity at site 1.

Site 2

Neither treatment, day, nor day*treatment interaction significantly affected the lateral to medial medullary cavity width at site 2 (Figure 124, Tables A-31 and B-123). The diameter of the medullary cavity did not change over the course of the trial. The two treatment groups did not differ from each other at any point during the trial.

Normalized data from the lateral to medial medullary cavity width at site 2 were also evaluated (Figure 125, Tables A-32 and B-124). Once again, there were no significant effects to the lateral to medial medullary cavity width at site 2 due to treatment, day, or a day*treatment interaction. The diameter of the medullary cavity did not change significantly during the research project. There was a different pattern of change in the two treatment groups. The Con group had a minor gain in medullary cavity width at day 64 followed by a minor loss back to day 0 values by day 128. The eST group had a larger, but also non-significant loss in medullary cavity width at day 64 that partially disappeared by day 128. There were no significant differences between the two treatment groups.

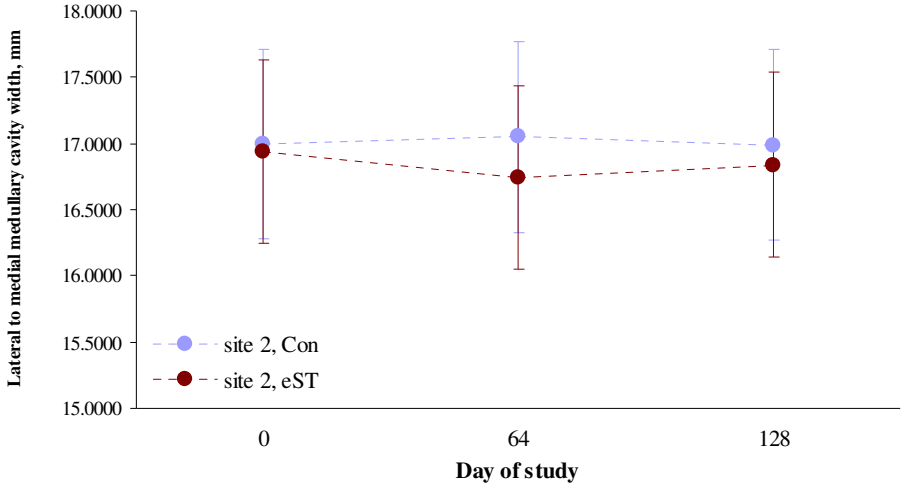


Figure 124. Lateral to medial medullary cavity width in mm, at site 2, vs day of study.

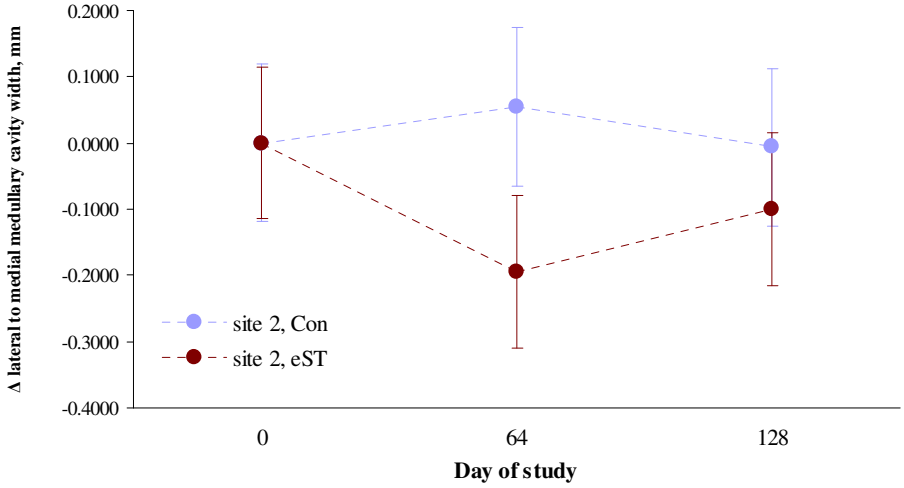


Figure 125. Change in lateral to medial medullary cavity width in mm, at site 2, vs day of study.

Site 3

The lateral to medial medullary cavity width at site 3 was not affected by treatment, day, or day*treatment interaction (Figure 126, Tables A-31 and B-125). No significant changes in the medullary width occurred overall or in the Con group. The eST group did not change from day 0 to day 64 or day 64 to day 128, but did have a trend (P=.08) for the decrease in width from day 0 to 128 to be statistical. The two treatment groups did not differ from each other.

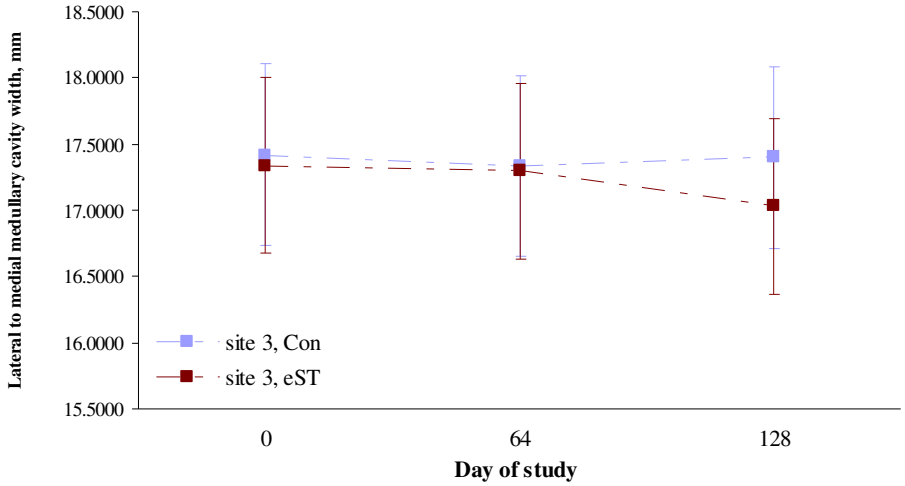


Figure 126. Lateral to medial medullary cavity width in mm, at site 3, vs day of study.

The data from the lateral to medial medullary cavity width at site 3 were normalized to day 0 values and re-evaluated to more closely examine any changes in medullary cavity width that may have occurred during the course of the experiment (Figure 127, Tables A-32 and B-126). The medullary cavity width was not significantly affected by day, treatment, or by any day*treatment interaction. The medullary cavity width did not vary significantly from day 0 to day 128. The Con group had a non-significant increase in the diameter of the medullary cavity from day 64 to day 128, while the eST group had

a non-significant decrease in the diameter of the medullary cavity during the same time period, and a trend for the decrease from day 0 to day 128 to be significant (P=.08). Consequently, the two groups differed on day 128, with the Con group having less of a decrease in medullary cavity width at site 3 than the eST group, but this difference was not of statistical importance.

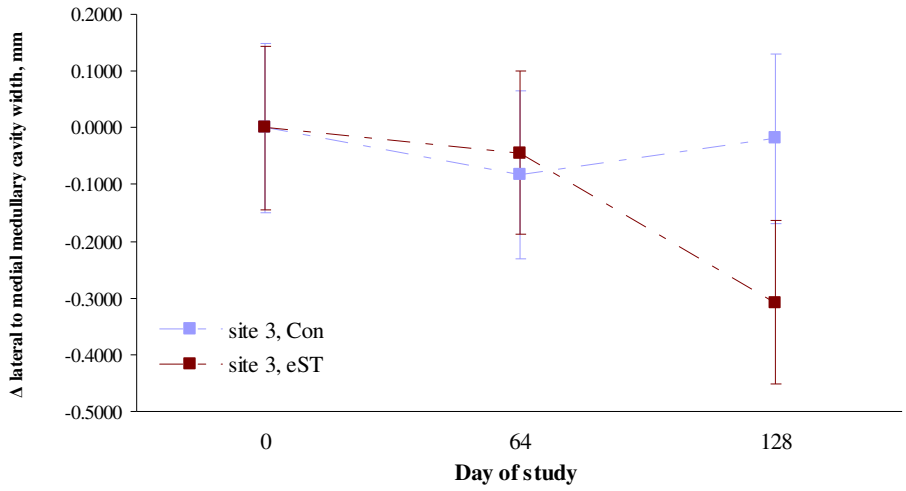


Figure 127. Change in lateral to medial medullary cavity width in mm, at site 3, vs day of study.

Site 4

There were no significant effects on the lateral to medial medullary cavity width at site 4 due to treatment, day, or day*treatment interaction (Figure 128, Tables A-31 and B-127). There was no change in the medullary cavity width from day 0 to day 128 in either the entire group of horses or in the Con treatment group. The eST treatment group had a significant decrease in medullary cavity width from day 0 to day 64 (P=.02), and a non-significant increase from day 64 to day 128. The two treatment groups did not significantly differ from each other on any of the sample days.

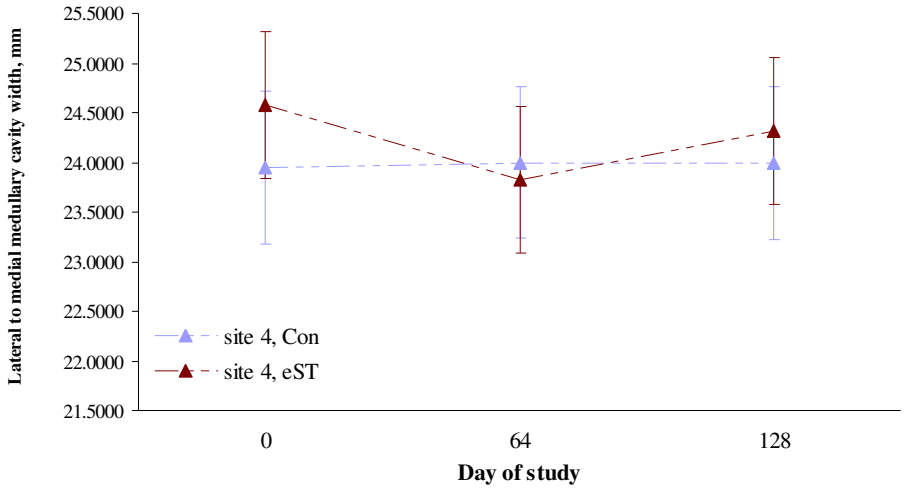


Figure 128. Lateral to medial medullary cavity width in mm, at site 4, vs day of study.

Day 0 values were subtracted from the lateral to medial medullary cavity width at site 4 to more closely examine any changes in the width of the medullary cavity over the time frame of the project and statistics were run on the transformed data (Figure 129, Tables A-32 and B-128). There were no significant effects due to day, treatment, or day*treatment interaction on the width of the medullary cavity at site 4. No significant changes in the diameter of the medullary cavity occurred from day 0 to day 128 in either the overall group of horses or in the Con treatment group. The eST treatment group had a significant decrease in the width of the medullary cavity from day 0 to day 64 (P=.02) followed by a non-significant increase in the width of the medullary cavity from day 64 to day 128. The two treatment groups differed from each other on day 64 (P=.04) due to the decrease in medullary cavity width that occurred in the eST treatment group. The difference between the two treatment groups decreased after day 64, and was not significant on day 128.

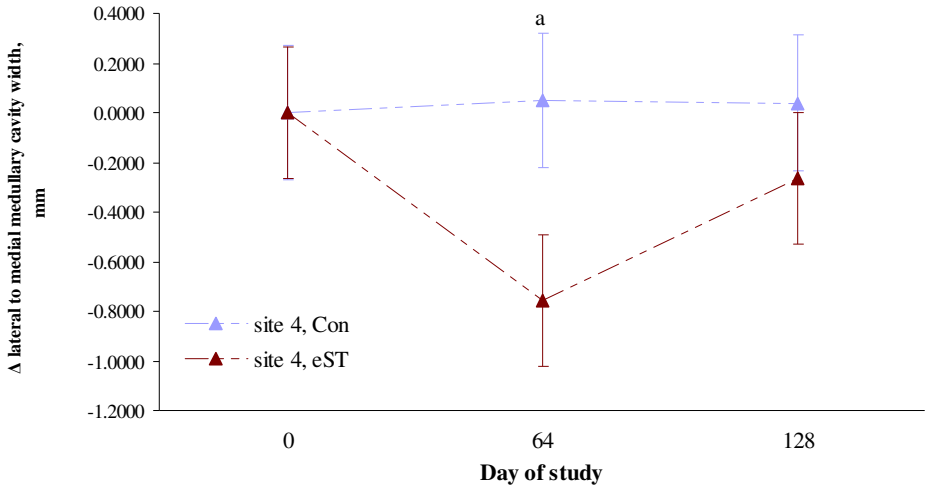


Figure 129. Change in lateral to medial medullary cavity width in mm, at site 4, vs day of study.
[a] Treatments differ (P≤.05).

Lateral to Medial Bone Diameter Micrometer Readings

Site 1

The lateral to medial bone diameter at site 1 had a trend to be affected by a day*treatment interaction (P=.09) but was not significantly affected by day or by treatment (Figure 130, Tables A-33 and B-129). There was no significant change in the bone diameter from day 0 to day 64 or from day 64 to day 128, but there was a trend (P=.09) for day 128 to be different from day 0. The Con group had a non-statistical increase in the bone diameter from day 0 to day 64 and a non-statistical decrease in bone diameter from day 64 to day 128. In contrast, the eST group had a non-statistical decrease in the bone diameter from day 0 to day 64 and a significant increase in bone diameter from day 64 to day 128 (P=.02). The eST group had a lateral to medial bone diameter at site 1 on day 128 that was significantly greater than on day 0 (P=.04). The Con group had a non-significantly greater bone diameter than the eST group on day 0 that increased and trended toward significance on day 64 (P=.09) and decreased back into non-significance on day 128.

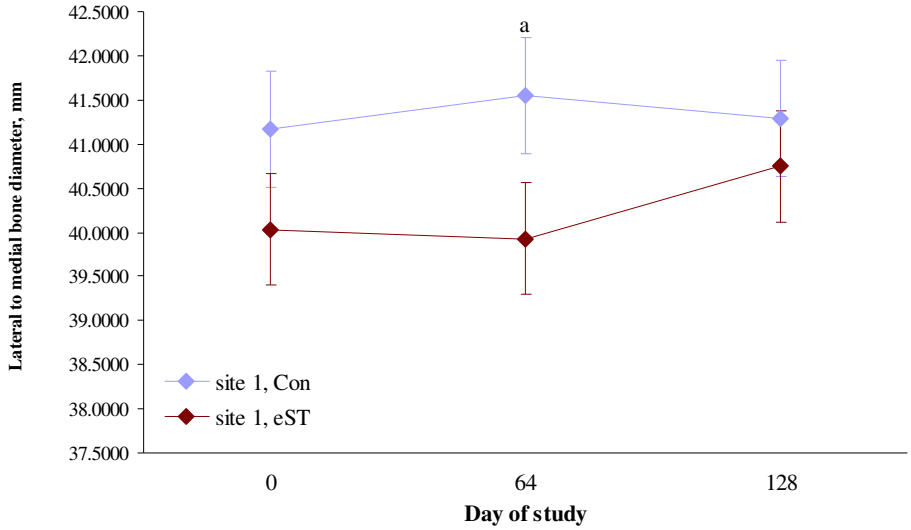


Figure 130. Lateral to medial bone diameter in mm, at site 1, vs day of study.
[a] Trend for treatments to differ (P≤.10).

The lateral to medial bone diameter at site 1 data were normalized to remove day 0 values and re-evaluated to better define any changes that occurred over the time span of this trial (Figure 131, Tables A-34 and B-130). There was a trend for a day*treatment interaction to affect the bone diameter (P=.09), but no treatment or day effect. A non-significant gain in bone diameter from day 0 to day 64 and from day 64 to day 128 resulted in a trend for the gain in bone diameter from day 0 to day 128 to be significant (P=.09). The Con group of horses had an increase in bone diameter from day 0 to day 64 and a decrease in bone diameter from day 64 to day 128, neither of which was significant. The eST group of horses had a contrasting pattern of change. The eST group of horses had a non-significant decrease in bone diameter from day 0 to day 64 and a significant increase in bone diameter from day 64 to day 128 (P=.02). The day 128 bone diameter in the eST group of horses was significantly greater than the day 0 value in the same group of horses (P=.04). The two treatment groups did not significantly differ from each other on the sample days.

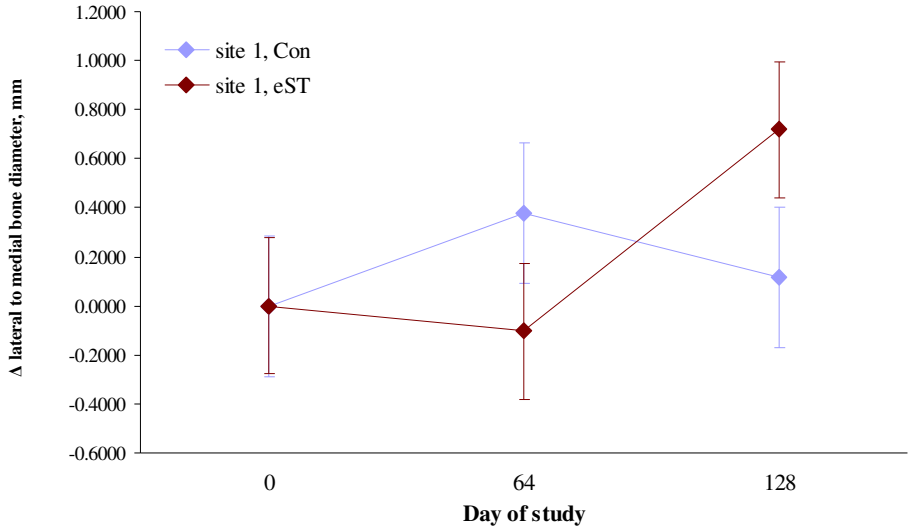


Figure 131. Change in lateral to medial bone diameter in mm, at site 1, vs day of study.

Site 2

The lateral to medial bone diameter at site 2 had a trend to be affected by day (P=.06) but was not significantly affected by treatment or by a day*treatment interaction (Figure 132, Tables A-33 and B-131). There was a non-significant increase in bone diameter in the lateral to medial direction from day 0 to day 64 and from day 64 to day 128. When day 0 was compared to day 128 the increase in bone diameter was significant (P=.02). Both treatment groups had the same pattern of change in bone diameter with non-significant increases from day 0 to day 64 and from day 64 to day 128, and with a trend for the increase from day 0 to day 128 to be significant in both the Con group (P=.100) and the eST group (P=.09). The Con group started the trial on day 0 with a non-statistically greater bone diameter than the eST group that continued through the end of the trial.

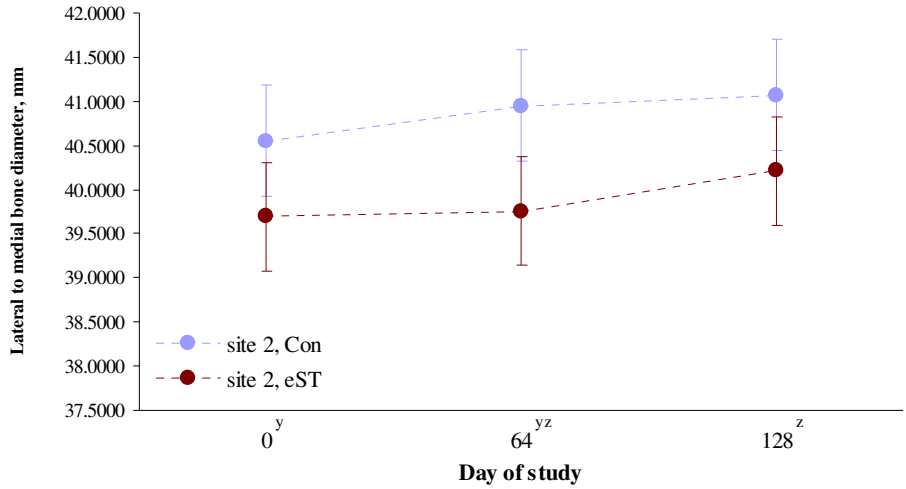


Figure 132. Lateral to medial bone diameter in mm, at site 2, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.01).

Data from the lateral to medial bone diameter at site 2 were normalized to day 0 values and statistics were ru-run to look at changes in the bone diameter that occurred during the research project (Figure 133, Tables A-34 and B-132). There was a trend for day to affect the bone diameter at site 2 (P=.06), but there were no significant affects due to treatment or to any day*treatment interaction. There was an increase in the lateral to medial bone diameter that was not significant from day 0 to day 64 or from day 64 to day 128, but was significant from day 0 to day 128 (P=.02). Neither of the two treatment groups had significant changes in the lateral to medial bone diameter at site 2 from day 0 to day 64 or from day 64 to day 128, but there was a trend for the increase in the bone diameter from day 0 to day 128 to be significant in both the Con group (P=.100) and the eST group (P=.09). The two treatment groups did not differ from each other statistically.

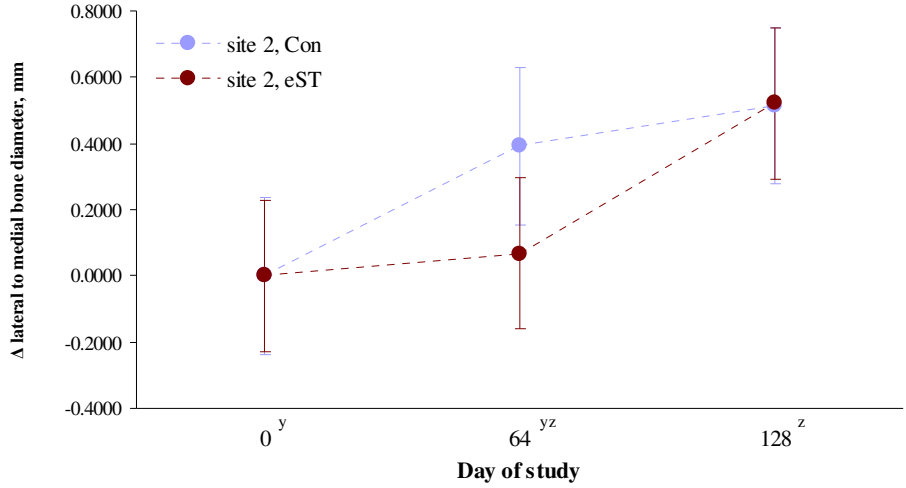


Figure 133. Change in lateral to medial bone diameter in mm, at site 2, vs day of study.
[yz] Days not sharing the same superscript differ (P≤.05).

Site 3

There were no significant effects caused by day, treatment, or a day*treatment interaction that affected the lateral to medial bone diameter at site 3 (Figure 134, Tables A-33 and B-133). There was no significant change in the bone diameter either overall in the entire number of horses or in the Con group of horses during the trial. The eST group of horses also had no significant change in bone diameter at site 3 from day 0 to day 64 or from day 64 to day 128, however there was a trend for the bone diameter on day 128 to be greater than on day 0 (P=.08). The non-significantly greater bone diameter on day 0 in the Con group as compared to the eST group continued through day 128.

Data from the lateral to medial bone diameter at site 3 were normalized by subtracting day 0 values, and the normalized data were analyzed statistically to evaluate changes in bone diameter that may have occurred during this experiment (Figure 135, Tables A-34 and B-134). The bone diameter at site 3 was not significantly affected by treatment, day, or day*treatment interaction. There were no significant changes in the bone diameter over the course of the project. The two treatment groups did not differ from each other on any of the sampled days.

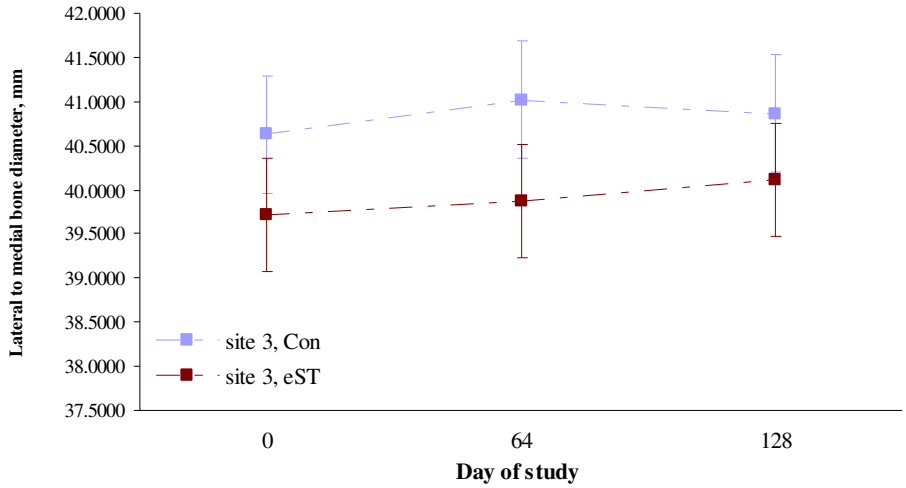


Figure 134. Lateral to medial bone diameter in mm, at site 3, vs day of study.

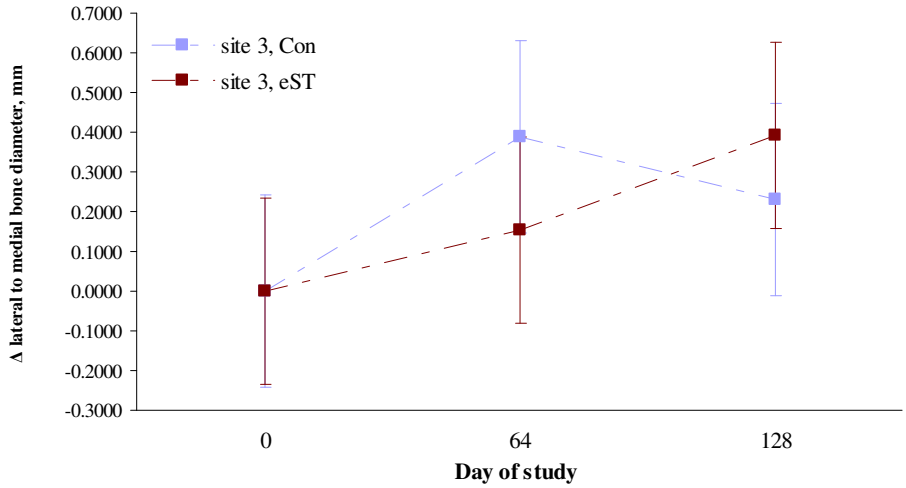


Figure 135. Change in lateral to medial bone diameter in mm, at site 3, vs day of study.

Site 4

The lateral to medial bone diameter at site 4 was not significantly affected by treatment, day, or any day*treatment interaction (Figure 136, Tables A-33 and B-135). There was an increase in the bone diameter at this site that had a trend to be significant from day 0 to day 128 (P=.07), but was not significant from day 0 to day 64 or from day 64 to day 128. The Con group did not have a statistical change in bone diameter. The eST group did not have a statistical change in bone diameter from day 0 to day 64, but an increase in the bone diameter after day 64 resulted in a trend for day 128 values to be greater than day 64 values (P=.08), and a significant difference between day 0 and day 128 (P=.02). The Con group had a larger bone diameter than the eST group for the duration of the trial, but this difference was not significant.

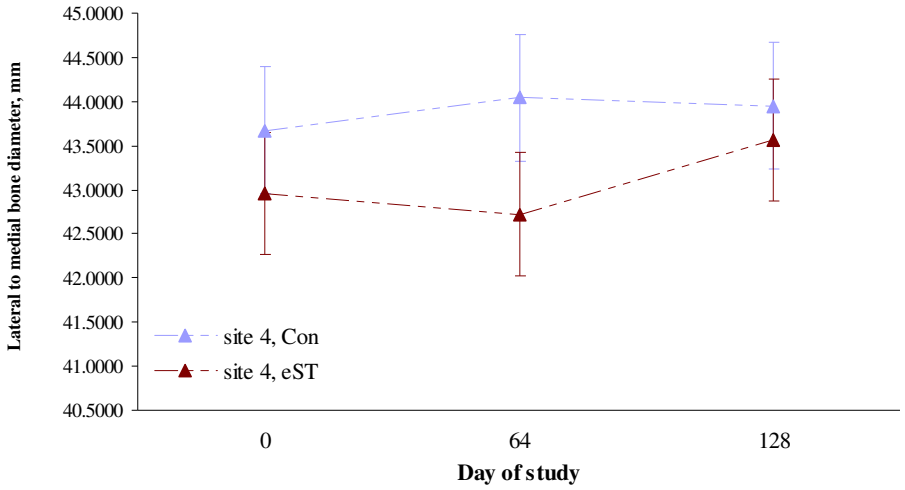


Figure 136. Lateral to medial bone diameter in mm, at site 4, vs day of study.

Data from the lateral to medial bone diameter at site 4 were normalized to day 0 values and statistics were re-run to better evaluate any changes in the width of the bone at site 4 over the duration of this project (Figure 137, Tables A-34 and B-136). The bone

diameter at this site was not significantly affected by day, treatment, or by any day*treatment interaction. The width of the bone did not change significantly from day 0 to day 64 or from day 64 to day 128, but the bone width did change enough for there to be a trend for day 128 values to be greater than day 0 values (P=.07). The Con group did not have a significant change in lateral to medial bone diameter at site 4 during this trial. The eST group had a non-significant decrease in bone diameter from day 0 to day 64 and a significant gain in the bone diameter from day 64 to day 128 (P=.02). In the eST group, day 128 had a trend to bone diameter that was greater than day 0 (P=.08). The two treatment groups did not differ from each other statistically.

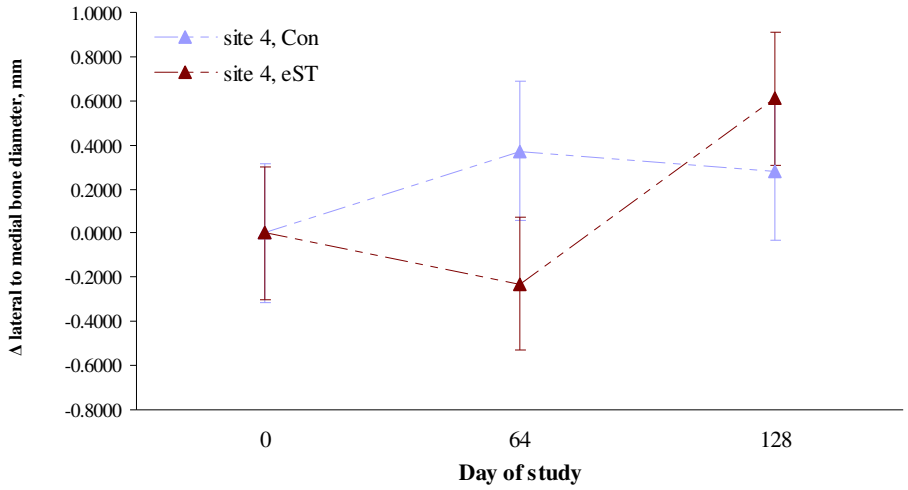


Figure 137. Change in lateral to medial bone diameter in mm, at site 4, vs day of study.

Bone Index Value of Micrometer Readings

The bone index value (Figure 138, Tables A-35 and B-137) was calculated at site 3 using the method of Larkin and Davies (1996). The bone index was evaluated at site 3 because this was the area Larkin and Davies (1996) evaluated and found to be an indicator of probablility of future shin soreness in racehorses. This author did not find

any published reports of bone index values at other sites, nor any correlation of the bone index values at other sites to probability of injury or shin soreness, and therefore did not calculate bone index values at the other sites (site 1, site 2, or site 4) for this project.

There was not a difference in the index value at site 3 from day 0 to day 64. On day 128 the index value was significantly greater than on day 0 (P<.0001) or on day 128 (P<.0001). The two treatment groups did not differ from each other in the calculated index value on day 0 or on day 64. By day 128 the two groups differed from each other non-significantly (P=.101) with the eST group having a larger index value than the Con group.

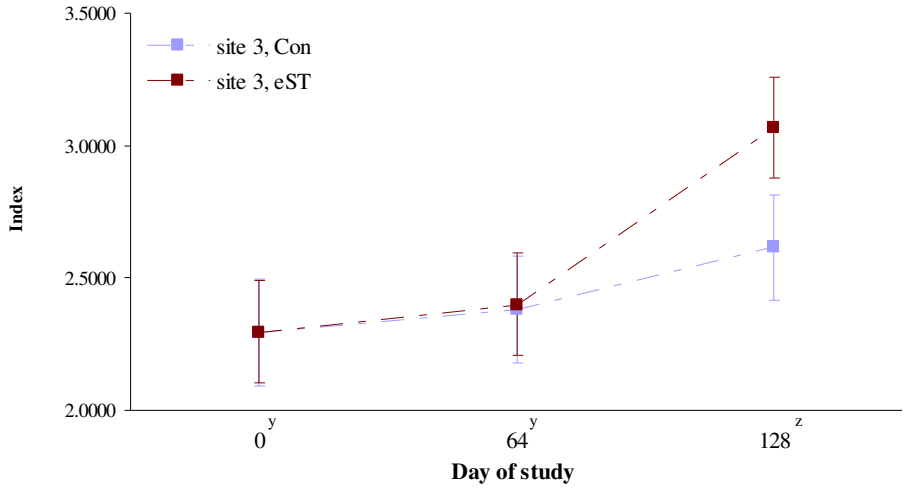


Figure 138. Computed bone index value of micrometer readings at site 3, vs day of study.
[yz] Days not sharing the same superscript differ (P<.0001).

The data were normalized to day 0 and statistics were re-run on the normalized data (Figure 139, Tables A-36 and B-138). There were no significant differences between day 0 and day 64, but there was an increase by day 128 that was significantly greater than day 0 or day 64 (both P<.0001). The two treatment groups did not differ from each other in the calculated normalized index value on day 0 or day 64. On day 128, the eST group

had a significantly greater increase in the index of bone value than did the Con group (P=.007).

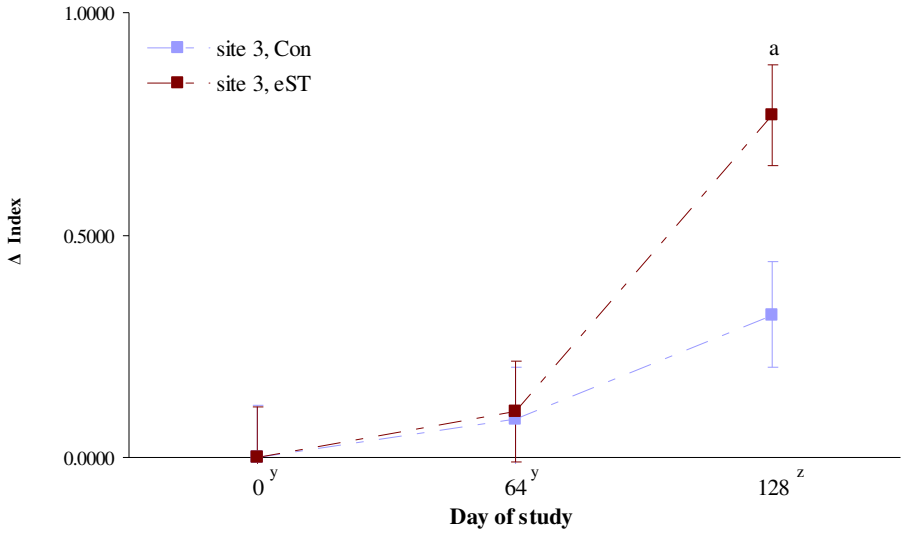


Figure 139. Normalized computed bone index value of micrometer readings at site 3, vs day of study.

[a] Treatments differ (P≤.01).

[yz] Days not sharing the same superscript differ (P<.0001).

DISCUSSION

During the course of this experiment there were no observed adverse reactions to the daily injections of eST. This is similar to what has been noted in other studies. Porcine growth hormone caused serious injection site reactions in horses, bovine growth hormone caused a mild adverse reaction in one of three horses to which it was administered, while no adverse reactions were seen with deep intermuscular injection of equine growth hormone to horses in a study conducted by De Kock et al. (2001). Bresegen, the manufacturer of the recombinant equine growth hormone used in this trial, does recommend deep intermuscular administration of their product to equines (as compared to subcutaneous injection of growth hormone, a frequent administration route of growth hormone to humans) as swelling and pain have been reported with subcutaneous injection of growth hormone in the horse. No significant increases in heart rate (and therefore no indication of significant pain) were associated with the actual injection of eST in six horses given up to five times the recommended dose of recombinant growth hormone in an experiment in Australia (Dart et al. 1998).

Site 4 micrometer readings were hard to obtain. At this location the trabecular bone merges with the cortical bone without a distinct margin between the two types of bone. Through repeated measurements it was possible to obtain two readings from site 4 that were within the exceptable margin of error for every radiograph. However, it is the opinion of this investigator that the site 4 measurements had a low repeateability and a much greater margin of error than did the other three sites that were evaluated in this trial. Additionally, the pattern of change over time at site 4 did not always correspond with patterns of change over time seen at site 1, site 2, and site 3. The thinner cortical bone and much more prevelant trabecular bone seen at site 4 that were present on day 0 and continued through the entire 128 day study, as well as the different patterns of change over time are likely the result of different stresses influencing the modeling of bone in this location as compared to the more mid-diaphasis locations of site 1, site 2, and site 3.

The two treatment groups differed in average body weight on day 0. Efforts were made to pair the animals based on sex and age, then one animal from each pair was randomly assigned to each treatment group. By random chance, more of the lighter weight animals ended up in one treatment group. The difference in body weight between the two treatment groups on day 0 was unfortunate, especially as bone development is greatly influenced by body weight. Normalizing the data to day 0 values helped to negate the diffences that existed between the two treatment groups at the start of the trial. However, it may be possible that some of the differences in bone growth seen between the treatment groups may be the result, at least in part, of the difference in bone development between the two groups at the start of the trial. Ideally the animal pairs would have been matched by body weight along with age and sex, but this would have required a larger group of animals. Since the horses used in this trial were loaned to us from various owners across the United States it was not possible to eliminate all the differences between the animal pairs.

Stress is based on amount of force applied and the rate with which it is applied. The force applied would have been greater in the heavier horses (the Con group) than in the lighter weight horses (the eST group) because of the difference in body weight. Therefore, the stress of the exercise regime was greater in the Con group at the start of the trial. The eST group of horses gained a significantly greater amount of weight during the trial than did the Con group (Sutfin, 2000), so as the trial progressed the stress of the exercise regime no longered differed between the two groups.

Strain is the amount of deformation caused by the stress applied to the bone. The strain would vary with the diameter and the density of the bone. Since strain gauges were not used in this experiment, it can not be determined if the two groups experienced the same amount of strain.

Somatotropin treatment did affect bone growth in a positive manner. The horses that were in the eST treatment group had a greater increase in bone mineral density as measured by total RBAE than did the horses in the Con group. By day 128 the two treatment groups differed in the change that occurred over the course of the experiment

in total RBAE at site 1, site 2, and site 3, with a greater gain in total RBAE in the eST group than in the Con group of horses.

Bone that has a greater mineral density can withstand greater compression (Loveridge, 1999). Increase in mineral density increases the strength of bone by affecting material properties of the bone. When the bone becomes highly mineralized (mineral content greater than 60% of ash weight), however, it is easier for microfractures to propagate through the bone (Loveridge, 1999). The bones of the horses in this study were not examined in vitro, so the % mineral content is not known. There were not any in vivo indications of microfractures, as the horses did not display signs of shin soreness or other lamenesses except for the afore noted stone bruising of the feet exhibited by two horses, and the pre-existing bone cyst exhibited by one horse (all three of which were removed from the study). Thus, the increase in total bone mineral density of the eST horses in this study appears to correspond with a positive increase in bone strength.

In the medial cortex and the dorsal cortex, at all four sites, the gain in RBAE over the 128 days was greater in the eST group than the Con group, though none of the overall treatment differences were significant. In the palmar cortex, the opposite occurred, where the gain in RBAE over the 128 days was non-significantly greater in the Con group than in the eST group. In the lateral cortex, the RBAE change was no different between the two treatment groups by day 128.

The dorsomedial part of the third metacarpus of the horse undergoes the greatest stress during exercise (Ordige, 1985). This is the area where the greatest increase in bone mineral density occurred in the eST group of horses, presumably because the eST treated horses were selectively depositing bone mineral in the area of greatest strain to decrease the exercise-induced deformation of the bone in that area. The increase in RBAE in the dorsal cortex and the medial cortex could be because of somatotrophin repartioning nutrients to increased bone growth throughout the animal. But, if that were the case, the eST group should have also exhibited greater bone mineral deposition in the lateral and palmar aspects of the third metacarpus than what was seen in the Con group. That was not the case. Instead, it appears that the eST group of horses selectively increased the bone mineral density of the third metacarpal in the areas that undergo the

greatest strain. This may be due to somatostatin treatment causing repartioning of the dietary nutrients to preferentially increase the amount of bone mineral that is deposited at the sites of greatest strain. Alternatively, this may be due to somatostatin treatment causing a reset of the "mechanostat" Frost has proposed (1987), causing the eST treated horses to remodel bone at a lower strain magnitude than the Con horses. If this is the case, the eST treated group of horses would have bone that was better able to withstand the rigors of training at any given stress level than non-treated horses.

The ratio of dorsal to palmar cortices was more dramatically increased in the eST treated horses than in the Con horses. No difference occurred between the two treatment groups in the amount of increase in the ratio of lateral to palmar cortices. The eST group had a non-significantly greater increase in the medial to lateral cortices and in the ratio of the medial to palmar cortices than did the Con group.

Treatment with somatotropin did positively influence the eST group to preferentially increase the bone mineral density of the cortices that undergo the greatest strain.

Bone strength is influenced not only by the material makeup of the bone, but also by the geometric structure of the bone. Bone that has a larger diameter has a greater moment of inertia, decreasing the strain the bone experiences at any given level of stress.

The eST treated horses had a greater increase in cross-sectional geometry of the bone as measured by micrometer readings of radiographs than did the Con horses. The increase in the width of the dorsal cortex was significantly greater in the eST group than in the Con group at all four of the measured sites by day 128. There was also a greater increase in the width of the medial cortex of the eST group than the Con group at all four sites by day 128, though this difference did not reach significance at two of the four sites. The eST group had a non-significantly greater increase in the width of the palmar cortex than did the Con group at all four of the measured sites. The two treatment groups did not differ in the amount of increase seen in the width of the lateral cortex.

The eST group actually had a decrease in the dorsal to palmar medulary cavity width at all four sites, while the Con group showed little change in the medulary cavity width. For there to be a decrease in medulary cavity width, the eST group of horses had to undergo deposition of new bone on the endosteal surface.

The two groups did not have a consitent pattern of change in the lateral to medial medulary cavity width.

The eST group had a significantly greater increase in the dorsal to palmar bone diameter at site 1, site 2, and site 3, and a non-significantly greater increase in dorsal to palmar bone diameter at site 4 than did the Con group. The eST group had a non-significantly greater increase in lateral to medial bone diameter than did the Con group at site 1, site 3, and site 4, with no difference between the two treatment groups in the gain in lateral to medial bone diameter at site 2.

In the cortices experiencing the greatest strain, the eST group selectively deposited more bone on the periosteal surface and/or endosteal surfaces and/or decreased the resorption of bone from the endosteal surface to result in a greater width of the cortical bone. These changes would positively influence the moment of inertia, thus decreasing the strain the eST horses experienced at a given level of stress. The Con group of horses experienced the same type of changes, with a potential increased moment of intertia resulting from positive changes in the bone geometry, but the changes were less pronounced in the Con group than in the eST group.

The micrometer readings from site 3 were used to calculate a bone index using the method reported by Larkin and Davies (1996). The bone index compares the width of cortical bone in relationship to the width of the medullary cavity, and compares the width of the dorsal cortical bone in relationship to the width of the palmar cortical bone. Horses that are depositing more bone in the dorsal cortex, which is the cortex that experiences the greatest amount of strain in young racehorses, would have a greater increase in the bone index than would horses that were not as selective in modeling their bones to best withstand the stress of race training. The significantly greater increase in the index in the eST treated horses than in the Con horses is yet another indication of the positive effects that eST treatment had on bone modeling in the treated horses.

Overall, the greater positive changes in bone density and bone geometry in the eST treatment group show that somatotropin treatment can positively influence bone modeling and/or remodeling in juvenile horses in race training. The greater question, in this investigator's opinion, is if these positive changes in bone modeling/remodeling

warrant consideration of somatotropin treatment of juvenile racehorses as a routine practice in the racehorse industry.

McKeever et al. (1998) looked at chronic administration of eST and its potential benefits to exercise performance. The aged mares in McKeever's study did not have a positive effect on aerobic capacity or on exercise performance. Rose (1998) states that "if there were a physiological mechanism for eST improving aerobic capacity and exercise tolerance, a positive effect should have been evident in older untrained mares just as much as in younger trained racehorses". It does not appear that eST has a direct affects on a horse's exercise tolerance, but potential benefits to the juvenile racehorse include increased growth, increased bone mineral density, earlier maturity, and preferential partitioning of nutrients to muscle and lean body mass.

The racehorse industry has looked for a way to test for the abuse of exogenous somatotropin administration in racehorses. Because of its normal pusitile secretion and its short half-life, somatotropin levels in an animal can not be determined based on a single blood sample. The exogenous somatotropin used in this study varies only slightly (one amino acid) from naturally occurring somatotropin. De Kock et al. (2001) investigated the possibility of using IGF-1 as a marker of eST administration and found the results promising. Noble et al. (2000) documented that IGF-1 levels rise significantly above naturally occurring IGF-1 levels in somatotropin treated racehorses, and that the increased IGF-1 levels can be detected based on a single blood sample. In that study the increased level of IGF-1 could be detected after the horses had been on exogenous somatotropin treatment for 15 days, and continued to remain significantly eleveated throughout somatotropin treatment. The levels of IGF-1 returned to normal 10 days after somatotropin treatment was discontinued. Price et al. (2000) have noted that pro-collegen peptide of type I collagen (PICP) a marker of bone formation, and cross-linked telopeptide of type I collagen (ICTP) a marker of bone resorption, were both significantly elevated in somatotropin treated horses, offering another means of detecting exogenous somaotropin administration via a blood test.

In this researcher's opinion, it is unlikely that racehorses will be allowed to compete while undergoing detectable levels of exogenous somatotropin treatment. Treatment of

juvenile racehorses in training, before they are raced competively and subjected to blood testing requirements, may become a standard use of somatotropin in the racehorse, as trainers are attempting to reduce the amount of bone injuries in racehorses. However, the question of the advisability of using eST on a routine basis can not be answered until research has been conducted to determine what effects discontinuing somatotropin treatment may have on bone mineral density and bone growth in the horse.

Future research needs to investigate any changes in bone mineral density and bone geometry that may occur in horses after chronic eST administration has been discontinued.

It may be that the positive changes in bone density and bone geometry that occurs in eST treated horses compared to control horses will continue to be apparent even after eST treatment has been discontinued. If the treated horses continue to show advantages in bone remodeling and modeling or at least do not lose the advantage that was gained during eST treatment, the long term benefits of eST treatment will be well worth the administration of the hormone.

Alternatively, and more likely in this researcher's opinion, with the cessation of eST treatement the advantage in bone growth seen with eST treatment may disappear in a short amount of time. This could occur due to the "mechanostat" being re-set to the pre-treatment level. In this senereo, the previously somatotropin treated horses would no longer remodel their bones until the strain levels of these bones reached the new set point. This would result in these horses soon adjusting to the bone mineral density and bone geometry that they would have had if they had not undergone somatotropin treatment. In this senereo, the eST treatment would not have long-term benefits, but short-term benefits may include these horses being able to adjust to the rigors of race horse training with fewer bone injuries than untreated horses.

CONCLUSIONS

No adverse side effects due to somatotropin treatment were observed during this study.

The somatotropin treated horses in this research project had a greater increase in the total bone mineral density of the third metacarpal at the four sites measured than did the control horses. The eST treated horses had a greater increase in the dorsal to palmar bone diameter than the control horses. The eST horses had a decrease in the dorsal to palmar medullary cavity width, which was not seen in the control horses. All of these changes show that eST treatment does cause changes in bone remodeling and modeling that are associated with increased bone strength. The positive changes in bone geometry are reflected in the significantly greater positive changes in the bone index in the eST treated horses than in the Con horses. The increase in bone strength decreases the expected incidence of bone injury in these horses.

This study did not continue to follow the long term changes in bone density and geometry in these horses after the eST treatment was discontinued. It is not known if these positive changes in bone strength were negated after the eST treatment was discontinued or if the gain in bone strength had a long term positive effect on the somatotropin treated group of horses.

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APPENDIX A

MEANS TABLES

Table A-1. Total RBAE in mm^2 Al equivalence (mean ± SE).

day	0	32	50	64	82	96	128
Site 1							
Con	606.30 ± 31.02 ^{xy}	610.46 ± 31.46 ^{xy}	597.73 ± 31.47 ^{x}	615.81 ± 31.02 ^{xy}	655.18 ± 31.47 ^{yz}	644.51 ± 31.02 ^{xyz}	670.84 ± 31.02 ^{z}
eST	535.18 ± 29.97 ^{w}	599.83 ± 29.97 ^{x}	538.11 ± 30.84 ^{wy}	583.17 ± 29.97 ^{yx}	628.40 ± 30.84 ^{xz}	629.49 ± 29.97 ^{x}	676.91 ± 29.97 ^{z}
Site 2							
Con	568.55 ± 29.24 ^{wx}	573.49 ± 29.64 ^{wxy}	559.03 ± 29.65 ^{w}	575.83 ± 29.24 ^{wxy}	615.83 ± 29.65 ^{yz}	606.57 ± 29.24 ^{xyz}	640.74 ± 29.24 ^{z}
eST	505.31 ± 28.25 ^{w}	567.99 ± 28.25 ^{xy}	505.99 ± 29.04 ^{w}	546.33 ± 28.25 ^{wx}	595.45 ± 29.04 ^{y}	591.77 ± 28.25 ^{y}	642.76 ± 28.25 ^{z}
Site 3							
Con	545.45 ± 28.24 ^{wx}	555.24 ± 28.65 ^{wxy}	536.15 ± 28.66 ^{w}	554.99 ± 28.24 ^{wxy}	594.74 ± 28.66 ^{yz}	587.76 ± 28.24 ^{xyz}	626.09 ± 28.24 ^{z}
eST	483.84 ± 27.28 ^{v}	552.38 ± 27.28 ^{wx}	485.18 ± 28.08 ^{vy}	527.98 ± 27.28 ^{wy}	579.22 ± 28.08 ^{x}	569.52 ± 27.28 ^{wx}	625.93 ± 27.28 ^{z}
Site 4							
Con	526.24 ± 34.62 ^{x}	543.09 ± 35.16 ^{xy}	524.48 ± 35.18 ^{x}	537.62 ± 34.62 ^{xy}	592.68 ± 35.18 ^{yz}	582.21 ± 34.62 ^{xyz}	639.40 ± 34.62 ^{z}
eST	456.95 ± 33.44 ^{v}	557.60 ± 33.44 ^{wx}	477.65 ± 34.52 ^{vy}	527.05 ± 33.44 ^{wy}	585.63 ± 34.52 ^{xz}	564.74 ± 33.44 ^{wx}	643.21 ± 33.44 ^{z}

^{vwxyz} Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-2. Normalized total RBAE in mm^2 Al equivalence (mean ± SE).

day	0	32	50	64	82	96	128
Site 1							
Con	0 ± 26.39 [xy]	3.21 ± 26.90 [xy]	-8.49 ± 26.91 [x]	9.52 ± 26.39 [xy]	48.96 ± 26.91[yz]	38.21 ± 26.39 [xyz]	64.55 ± 26.39 [a,z]
eST	0 ± 25.49 [w]	64.65 ± 25.49 [x]	4.65 ± 26.49 [wy]	47.99 ± 25.49 [xy]	94.95 ± 26.49 [xz]	94.31 ± 25.49 [x]	141.73 ± 25.49 [z]
Site 2							
Con	0 ± 25.15 [wx]	4.47 ± 25.63 [wxy]	-9.63 ± 25.64 [w]	12.88 ± 25.15 [wxy]	47.17 ± 25.64 [yz]	38.02 ± 25.15 [xyz]	72.18 ± 25.15 [b,z]
eST	0 ± 24.30 [w]	62.38 ± 24.30 [x]	2.04 ± 25.23 [wy]	46.98 ± 24.30 [xy]	91.50 ± 25.23 [x]	86.46 ± 24.30 [x]	137.46 ± 24.30 [z]
Site 3							
Con	0 ± 24.56 [wx]	9.04 ± 25.02 [b,wxy]	-9.36 ± 25.04 [w]	9.54 ± 24.56 [wxy]	49.23 ± 25.04 [yz]	42.32 ± 24.56 [xyz]	80.65 ± 24.56 [b,z]
eST	0 ± 23.73 [v]	68.54 ± 23.73 [wx]	2.82 ± 24.64 [vy]	44.13 ± 23.73 [wy]	96.85 ± 24.64 [x]	85.67 ± 23.73 [wx]	142.08 ± 23.73 [z]
Site 4							
Con	0 ± 32.90 [x]	15.93 ± 33.48 [b,xy]	-1.99 ± 33.49 [x]	11.37 ± 32.90 [xy]	66.21 ± 33.49 [yz]	55.96 ± 32.90 [xyz]	113.16 ± 32.90 [z]
eST	0 ± 31.79 [v]	100.65 ± 31.79 [wx]	22.70 ± 32.91 [vy]	70.10 ± 31.79 [wy]	130.68 ± 32.91 [xz]	107.79 ± 31.79 [wx]	186.44 ± 31.79 [z]

[a] Treatments differ ($p \leq .05$).

[b] Trend for treatments to differ ($p \leq .10$).

[vwxyz] Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-3. Dorsal RBAE in mm Al equivalence (mean ± SE).

day	0	32	50	64	82	96	128
Site 1							
Con	18.15 ± 0.25 [wxy]	17.84 ± 0.25 [w]	18.04 ± 0.25 [a,wx]	18.02 ± 0.25 [wx]	18.37 ± 0.25 [xyz]	18.47 ± 0.25 [yz]	18.69 ± 0.25 [z]
eST	17.47 ± 0.24 [wx]	17.28 ± 0.24 [w]	17.27 ± 0.25 [w]	17.67 ± 0.24 [xy]	18.04 ± 0.25 [z]	17.90 ± 0.24 [yz]	18.22 ± 0.24 [z]
Site 2							
Con	17.53 ± 0.24 [vwx]	17.09 ± 0.24 [y]	17.31 ± 0.24 [vwy]	17.24 ± 0.24 [wy]	17.67 ± 0.24 [vxz]	17.71 ± 0.24 [xz]	16.90 ± 0.24 [z]
eST	16.90 ± 0.23 [x]	16.68 ± 0.23 [x]	16.64 ± 0.23 [x]	16.95 ± 0.23 [xy]	17.32 ± 0.23 [z]	17.24 ± 0.23 [yz]	17.41 ± 0.23 [z]
Site 3							
Con	17.19 ± 0.24 [wx]	16.76 ± 0.24 [y]	16.94 ± 0.24 [wy]	16.95 ± 0.24 [wy]	17.34 ± 0.24 [xz]	17.31 ± 0.24 [wxz]	17.56 ± 0.24 [z]
eST	16.53 ± 0.23 [wx]	16.28 ± 0.23 [w]	16.38 ± 0.24 [w]	16.59 ± 0.23 [wxy]	16.92 ± 0.24 [yz]	16.83 ± 0.23 [xyz]	17.09 ± 0.23 [z]
Site 4							
Con	15.96 ± 0.26 [wxy]	15.39 ± 0.27 [z]	15.55 ± 0.26 [wz]	15.73 ± 0.26 [wxz]	15.92 ± 0.26 [wxy]	16.05 ± 0.26 [xy]	16.33 ± 0.26 [y]
eST	15.46 ± 0.25 [xy]	15.28 ± 0.25 [x]	15.47 ± 0.26 [xy]	15.52 ± 0.25 [xy]	15.91 ± 0.26 [yz]	15.80 ± 0.25 [yz]	16.15 ± 0.25 [z]

[a] Treatments differ ($p \leq .05$).

[vwxyz] Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-4. Normalized dorsal RBAE in mm Al equivalence (mean ± SE).

day	0	32	50	64	82	196	128
Site 1							
Con	0 ± 0.19 ^{wxy}	-0.31 ± 0.20 ^{w}	-0.11 ± 0.19 ^{wx}	-0.13 ± 0.19 ^{wx}	0.22 ± 0.19 ^{xyz}	0.32 ± 0.19 ^{yz}	0.55 ± 0.19 ^{z}
eST	0 ± 0.18 ^{wx}	-0.11 ± 0.18 ^{w}	-0.19 ± 0.19 ^{w}	0.20 ± 0.18 ^{xy}	0.58 ± 0.19 ^{z}	0.44 ± 0.18 ^{yz}	0.75 ± 0.18 ^{z}
Site 2							
Con	0 ± 0.18 ^{vwx}	-0.45 ± 0.19 ^{y}	-0.22 ± 0.18 ^{vwy}	-0.29 ± 0.18 ^{vy}	0.14 ± 0.18 ^{wxz}	0.18 ± 0.18 ^{xz}	0.35 ± 0.18 ^{z}
eST	0 ± 0.17 ^{x}	-0.22 ± 0.17 ^{x}	-0.25 ± 0.18 ^{x}	0.06 ± 0.17 ^{xy}	0.43 ± 0.18 ^{z}	0.34 ± 0.17 ^{yz}	0.51 ± 0.17 ^{z}
Site 3							
Con	0 ± 0.19 ^{wx}	-0.43 ± 0.19 ^{y}	-0.25 ± 0.19 ^{wy}	-0.25 ± 0.19 ^{wy}	0.15 ± 0.19 ^{xz}	0.01 ± 0.19 ^{wxz}	0.36 ± 0.19 ^{z}
eST	0 ± 0.18 ^{wx}	-0.25 ± 0.18 ^{w}	-0.14 ± 0.19 ^{w}	0.06 ± 0.18 ^{wxy}	0.40 ± 0.19 ^{yz}	0.30 ± 0.18 ^{xyz}	0.56 ± 0.18 ^{z}
Site 4							
Con	0 ± 0.22 ^{wxy}	-0.57 ± 0.23 ^{z}	-0.41 ± 0.22 ^{wz}	-0.23 ± 0.22 ^{xyz}	-0.04 ± 0.22 ^{wxy}	0.10 ± 0.22 ^{xy}	0.37 ± 0.22 ^{y}
eST	0 ± 0.21 ^{wx}	-0.18 ± 0.21 ^{w}	0.02 ± 0.22 ^{wxy}	0.06 ± 0.21 ^{wxy}	0.46 ± 0.22 ^{yz}	0.34 ± 0.21 ^{xyz}	0.68 ± 0.21 ^{z}

^{vwxyz} Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-5. Lateral RBAE in mm Al equivalence (mean ± SE).

day	0	32	50	64	82	96	128
Site 1							
Con	19.17 ± 0.44 ^{y}	19.74 ± 0.45 ^{y}	19.14 ± 0.45 ^{y}	19.53 ± 0.44 ^{y}	19.65 ± 0.45 ^{y}	19.78 ± 0.44 ^{y}	20.83 ± 0.44 ^{z}
eST	18.09 ± 0.42 ^{x}	18.98 ± 0.42 ^{xy}	18.81 ± 0.44 ^{x}	19.07 ± 0.42 ^{xy}	19.36 ± 0.44 ^{xy}	19.66 ± 0.42 ^{yz}	20.38 ± 0.42 ^{z}
Site 2							
Con	19.14 ± 0.47 ^{wx}	19.73 ± 0.48 ^{wxy}	18.84 ± 0.48 ^{w}	19.58 ± 0.47 ^{wx}	19.68 ± 0.48 ^{wx}	19.83 ± 0.47 ^{xy}	21.07 ± 0.47 ^{z}
eST	18.65 ± 0.46 ^{x}	19.10 ± 0.46 ^{xy}	18.73 ± 0.47 ^{x}	19.04 ± 0.46 ^{xy}	19.44 ± 0.47 ^{xy}	19.68 ± 0.46 ^{y}	20.59 ± 0.46 ^{z}
Site 3							
Con	18.87 ± 0.49 ^{xy}	19.49 ± 0.50 ^{x}	18.48 ± 0.50 ^{y}	19.23 ± 0.49 ^{xy}	19.25 ± 0.50 ^{xy}	19.54 ± 0.49 ^{x}	20.97 ± 0.49 ^{z}
eST	18.38 ± 0.47 ^{x}	18.83 ± 0.47 ^{xy}	18.35 ± 0.49 ^{x}	18.57 ± 0.47 ^{xy}	19.29 ± 0.49 ^{xy}	19.41 ± 0.47 ^{y}	20.45 ± 0.47 ^{z}
Site 4							
Con	16.89 ± 0.44 ^{x}	17.54 ± 0.45 ^{xyz}	17.00 ± 0.45 ^{xy}	17.29 ± 0.44 ^{xy}	17.20 ± 0.45 ^{xy}	17.76 ± 0.44 ^{yz}	19.37 ± 0.44 ^{z}
eST	16.34 ± 0.43 ^{w}	16.96 ± 0.43 ^{wxy}	16.71 ± 0.44 ^{wx}	17.04 ± 0.43 ^{wxy}	17.33 ± 0.44 ^{xy}	17.63 ± 0.43 ^{y}	18.67 ± 0.43 ^{z}

^{wxyz} Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-6. Normalized lateral RBAE in mm Al equivalence (mean ± SE).

day	0	32	50	64	82	96	128
Site 1							
Con	0 ± 0.36 ^{y}	0.55 ± 0.37 ^{y}	-0.04 ± 0.37 ^{y}	0.36 ± 0.36 ^{y}	0.47 ± 0.37 ^{y}	0.61 ± 0.36 ^{y}	1.66 ± 0.36 ^{z}
eST	0 ± 0.35 ^{x}	0.17 ± 0.35 ^{xy}	-0.01 ± 0.37 ^{x}	0.27 ± 0.35 ^{xy}	0.54 ± 0.37 ^{xy}	0.86 ± 0.35 ^{yz}	1.57 ± 0.35 ^{z}
Site 2							
Con	0 ± 0.41 ^{xy}	0.57 ± 0.42 ^{xy}	-0.32 ± 0.42 ^{y}	0.44 ± 0.41 ^{xy}	0.51 ± 0.42 ^{xy}	0.69 ± 0.41 ^{x}	1.94 ± 0.41 ^{z}
eST	0 ± 0.40 ^{w}	0.44 ± 0.40 ^{wx}	0.07 ± 0.42 ^{w}	0.39 ± 0.40 ^{wx}	0.78 ± 0.42 ^{wxy}	1.03 ± 0.40 ^{y}	1.94 ± 0.40 ^{z}
Site 3							
Con	0 ± 0.43 ^{xy}	0.59 ± 0.44 ^{x}	-0.43 ± 0.44 ^{y}	0.35 ± 0.43 ^{xy}	0.35 ± 0.44 ^{xy}	0.67 ± 0.43 ^{x}	2.10 ± 0.43 ^{z}
eST	0 ± 0.42 ^{x}	0.45 ± 0.42 ^{xy}	-0.04 ± 0.44 ^{x}	0.19 ± 0.42 ^{xy}	0.90 ± 0.44 ^{xy}	1.03 ± 0.42 ^{y}	2.07 ± 0.42 ^{z}
Site 4							
Con	0 ± 0.41 ^{x}	0.63 ± 0.42 ^{xy}	0.08 ± 0.42 ^{xy}	0.40 ± 0.41 ^{xy}	0.27 ± 0.42 ^{xy}	0.87 ± 0.41 ^{y}	2.48 ± 0.41 ^{z}
eST	0 ± 0.39 ^{w}	0.62 ± 0.39 ^{wxy}	0.38 ± 0.41 ^{wx}	0.70 ± 0.39 ^{wxy}	1.00 ± 0.41 ^{xy}	1.29 ± 0.39 ^{y}	2.53 ± 0.39 ^{z}

^{wxyz} Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-7. Medial RBAE in mm Al equivalence (mean ± SE).

day	0	32	50	64	82	96	128
Site 1							
Con	20.51 ± 0.38 ^{v}	21.07 ± 0.39 ^{vwx}	20.99 ± 0.39 ^{vw}	21.27 ± 0.38 ^{wxy}	21.91 ± 0.39 ^{yz}	21.68 ± 0.38 ^{xy}	22.43 ± 0.38 ^{z}
eST	19.79 ± 0.37 ^{w}	20.56 ± 0.37 ^{x}	20.82 ± 0.38 ^{x}	21.16 ± 0.37 ^{xy}	21.70 ± 0.38 ^{y}	21.62 ± 0.37 ^{y}	22.39 ± 0.37 ^{z}
Site 2							
Con	20.66 ± 0.38 ^{v}	21.25 ± 0.39 ^{vwx}	21.16 ± 0.39 ^{vw}	21.50 ± 0.38 ^{wxy}	22.12 ± 0.39 ^{yz}	21.90 ± 0.38 ^{y}	22.72 ± 0.38 ^{z}
eST	19.97 ± 0.37 ^{w}	20.81 ± 0.37 ^{x}	21.06 ± 0.38 ^{x}	21.34 ± 0.37 ^{xy}	21.84 ± 0.38 ^{y}	21.84 ± 0.37 ^{y}	22.55 ± 0.37 ^{z}
Site 3							
Con	20.46 ± 0.39 ^{v}	21.12 ± 0.39 ^{vwx}	21.02 ± 0.39 ^{vw}	21.25 ± 0.39 ^{wxy}	21.92 ± 0.39 ^{yz}	21.73 ± 0.39 ^{xy}	22.60 ± 0.39 ^{z}
eST	19.90 ± 0.37 ^{v}	20.67 ± 0.37 ^{w}	20.88 ± 0.39 ^{wx}	21.25 ± 0.37 ^{wxy}	21.65 ± 0.39 ^{y}	21.53 ± 0.37 ^{xy}	22.40 ± 0.37 ^{z}
Site 4							
Con	18.59 ± 0.43 ^{x}	19.27 ± 0.44 ^{xy}	19.41 ± 0.44 ^{y}	19.29 ± 0.43 ^{y}	19.67 ± 0.44 ^{y}	19.75 ± 0.43 ^{y}	20.86 ± 0.43 ^{z}
eST	18.02 ± 0.42 ^{w}	18.87 ± 0.42 ^{x}	19.17 ± 0.43 ^{x}	19.28 ± 0.42 ^{xy}	19.99 ± 0.43 ^{yz}	19.42 ± 0.42 ^{xy}	20.48 ± 0.42 ^{z}

^{vwxyz} Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-8. Normalized medial RBAE in mm Al equivalence (mean ± SE).

day	0	32	50	64	82	96	128
Site 1							
Con	0 ± 0.37 ^{v}	0.55 ± 0.38 ^{vwx}	0.46 ± 0.38 ^{vw}	0.76 ± 0.37 ^{wxy}	1.39 ± 0.38 ^{yz}	1.17 ± 0.37 ^{xy}	1.92 ± 0.37 ^{z}
eST	0 ± 0.36 ^{v}	0.77 ± 0.36 ^{w}	1.05 ± 0.37 ^{wx}	1.37 ± 0.36 ^{xy}	1.93 ± 0.37 ^{yz}	1.83 ± 0.36 ^{y}	2.60 ± 0.36 ^{z}
Site 2							
Con	0 ± 0.38 ^{v}	0.58 ± 0.38 ^{vwx}	0.45 ± 0.38 ^{vw}	0.84 ± 0.38 ^{wxy}	1.44 ± 0.38 ^{yz}	1.24 ± 0.38 ^{xy}	2.06 ± 0.38 ^{z}
eST	0 ± 0.36 ^{w}	0.84 ± 0.36 ^{x}	1.11 ± 0.38 ^{x}	1.37 ± 0.36 ^{xy}	1.89 ± 0.38 ^{y}	1.87 ± 0.36 ^{y}	2.58 ± 0.36 ^{z}
Site 3							
Con	0 ± 0.39 ^{v}	0.65 ± 0.40 ^{vwx}	0.54 ± 0.40 ^{vw}	0.79 ± 0.39 ^{wxy}	1.44 ± 0.40 ^{yz}	1.27 ± 0.39 ^{xy}	2.14 ± 0.39 ^{z}
eST	0 ± 0.38 ^{v}	0.77 ± 0.38 ^{w}	1.00 ± 0.39 ^{wx}	1.34 ± 0.38 ^{wxy}	1.77 ± 0.39 ^{y}	1.62 ± 0.38 ^{xy}	2.50 ± 0.38 ^{z}
Site 4							
Con	0 ± 0.40 ^{x}	0.68 ± 0.41 ^{xy}	0.82 ± 0.41 ^{y}	0.71 ± 0.40 ^{xy}	1.08 ± 0.41 ^{y}	1.17 ± 0.40 ^{y}	2.28 ± 0.40 ^{z}
eST	0 ± 0.39 ^{w}	0.84 ± 0.39 ^{x}	1.17 ± 0.40 ^{x}	1.26 ± 0.39 ^{xy}	1.99 ± 0.40 ^{yz}	1.40 ± 0.39 ^{xy}	2.69 ± 0.39 ^{z}

^{vwxyz} Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-9. Palmar RBAE in mm Al equivalence (mean ± SE).

day	0	32	50	64	82	96	128
Site 1							
Con	15.69 ± 0.24 ^{y}	15.14 ± 0.26 ^{z}	15.40 ± 0.25 ^{yz}	15.61 ± 0.24 ^{yz}	15.66 ± 0.25 ^{y}	15.82 ± 0.24 ^{y}	15.83 ± 0.24 ^{y}
eST	15.44 ± 0.23 ^{xyz}	15.04 ± 0.23 ^{xy}	14.99 ± 0.24 ^{x}	15.26 ± 0.23 ^{xyz}	15.48 ± 0.24 ^{yz}	15.36 ± 0.23 ^{xyz}	15.55 ± 0.23 ^{z}
Site 2							
Con	14.66 ± 0.24 ^{xy}	14.21 ± 0.25 ^{xz}	14.17 ± 0.25 ^{z}	14.47 ± 0.24 ^{xyz}	14.56 ± 0.25 ^{xyz}	14.68 ± 0.24 ^{xy}	14.77 ± 0.24 ^{y}
eST	14.50 ± 0.24 ^{x}	14.05 ± 0.24 ^{yz}	13.90 ± 0.26 ^{y}	14.23 ± 0.24 ^{xyz}	14.41 ± 0.25 ^{xz}	14.36 ± 0.24 ^{xyz}	14.35 ± 0.24 ^{xyz}
Site 3							
Con	14.21 ± 0.26 ^{xyz}	13.75 ± 0.27 ^{xy}	13.73 ± 0.27 ^{x}	13.91 ± 0.27 ^{xyz}	14.06 ± 0.27 ^{xyz}	14.21 ± 0.26 ^{yz}	14.25 ± 0.26 ^{z}
eST	14.06 ± 0.26 ^{y}	13.61 ± 0.25 ^{z}	13.49 ± 0.28 ^{z}	13.76 ± 0.26 ^{yz}	13.99 ± 0.27 ^{yz}	13.89 ± 0.25 ^{yz}	13.87 ± 0.26 ^{yz}
Site 4							
Con	13.71 ± 0.29 ^{yz}	13.23 ± 0.30 ^{y}	13.43 ± 0.31 ^{yz}	13.57 ± 0.30 ^{yz}	13.58 ± 0.30 ^{yz}	13.93 ± 0.29 ^{z}	13.91 ± 0.30 ^{z}
eST	13.44 ± 0.28 ^{z}	13.04 ± 0.28 ^{z}	13.12 ± 0.31 ^{z}	13.15 ± 0.28 ^{z}	13.44 ± 0.30 ^{z}	13.33 ± 0.28 ^{z}	13.38 ± 0.28 ^{z}

^{vwxyz} Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-10. Normalized palmar RBAE in mm Al equivalence (mean ± SE).

day	0	32	50	64	82	96	128
Site 1							
Con	0 ± 0.22 ^{y}	-0.55 ± 0.24 ^{z}	-0.30 ± 0.23 ^{yz}	-0.08 ± 0.22 ^{y}	-0.04 ± 0.23 ^{y}	0.13 ± 0.22 ^{y}	0.14 ± 0.22 ^{y}
eST	0 ± 0.22 ^{xyz}	-0.40 ± 0.22 ^{xy}	-0.45 ± 0.23 ^{x}	-0.18 ± 0.22 ^{xyz}	0.04 ± 0.23 ^{yz}	-0.08 ± 0.22 ^{xyz}	0.11 ± 0.22 ^{z}
Site 2							
Con	0 ± 0.21 ^{xy}	-0.45 ± 0.23 ^{xz}	-0.50 ± 0.22 ^{z}	-0.18 ± 0.21 ^{xyz}	-0.12 ± 0.22 ^{xyz}	0.03 ± 0.21 ^{y}	0.11 ± 0.21 ^{y}
eST	0 ± 0.21 ^{x}	-0.45 ± 0.21 ^{yz}	-0.63 ± 0.23 ^{y}	-0.27 ± 0.22 ^{xyz}	-0.10 ± 0.22 ^{xz}	-0.14 ± 0.21 ^{xz}	-0.15 ± 0.21 ^{xz}
Site 3							
Con	0 ± 0.21 ^{x}	-0.46 ± 0.22 ^{xy}	-0.50 ± 0.22 ^{y}	-0.32 ± 0.22 ^{xyz}	-0.17 ± 0.22 ^{xyz}	-0.01 ± 0.21 ^{xz}	0.04 ± 0.21 ^{z}
eST	0 ± 0.20 ^{y}	-0.40 ± 0.21 ^{yz}	-0.60 ± 0.24 ^{z}	-0.30 ± 0.21 ^{yz}	-0.11 ± 0.23 ^{yz}	-0.20 ± 0.21 ^{yz}	-0.19 ± 0.21 ^{yz}
Site 4							
Con	0 ± 0.24 ^{yz}	-0.49 ± 0.26 ^{y}	-0.26 ± 0.27 ^{yz}	-0.17 ± 0.25 ^{yz}	-0.19 ± 0.25 ^{yz}	0.24 ± 0.25 ^{z}	0.22 ± 0.25 ^{z}
eST	0 ± 0.23 ^{z}	-0.39 ± 0.24 ^{z}	-0.31 ± 0.27 ^{z}	-0.29 ± 0.24 ^{z}	0.02 ± 0.26 ^{z}	-0.12 ± 0.24 ^{z}	-0.04 ± 0.24 ^{z}

^{xyz} Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-11. Dorsal by palmar RBAE in mm Al equivalence (mean ± SE).

day	0	32	50	64	82	96	128
Site 1							
Con	1.16 ± 0.01 [xy]	1.18 ± 0.01 [a,xz]	1.17 ± 0.01 [xyz]	1.16 ± 0.01 [y]	1.18 ± 0.01 [xyz]	1.17 ± 0.01 [xyz]	1.18 ± 0.01 [z]
eST	1.13 ± 0.01 [y]	1.15 ± 0.01 [yz]	1.15 ± 0.01 [yz]	1.16 ± 0.01 [z]	1.17 ± 0.01 [z]	1.17 ± 0.01 [z]	1.17 ± 0.01 [z]
Site 2							
Con	1.20 ± 0.01 [yz]	1.21 ± 0.01 [yz]	1.22 ± 0.01 [y]	1.19 ± 0.01 [z]	1.22 ± 0.01 [yz]	1.21 ± 0.01 [yz]	1.21 ± 0.01 [yz]
eST	1.17 ± 0.01 [x]	1.19 ± 0.01 [xy]	1.20 ± 0.01 [yz]	1.20 ± 0.01 [yz]	1.20 ± 0.01 [yz]	1.20 ± 0.01 [yz]	1.21 ± 0.01 [z]
Site 3							
Con	1.21 ± 0.02 [z]	1.22 ± 0.02 [z]	1.24 ± 0.02 [z]	1.22 ± 0.02 [z]	1.24 ± 0.02 [z]	1.22 ± 0.02 [z]	1.24 ± 0.02 [z]
eST	1.18 ± 0.02 [x]	1.20 ± 0.02 [xy]	1.23 ± 0.02 [yz]	1.21 ± 0.02 [yz]	1.21 ± 0.02 [yz]	1.21 ± 0.02 [yz]	1.23 ± 0.02 [z]
Site 4							
Con	1.17 ± 0.02 [z]	1.17 ± 0.02 [z]	1.17 ± 0.02 [z]	1.16 ± 0.02 [z]	1.18 ± 0.02 [z]	1.15 ± 0.02 [z]	1.18 ± 0.02 [z]
eST	1.16 ± 0.02 [x]	1.18 ± 0.02 [xy]	1.19 ± 0.02 [xyz]	1.19 ± 0.02 [xyz]	1.19 ± 0.02 [xyz]	1.19 ± 0.02 [yz]	1.21 ± 0.02 [z]

[a] Trend for treatments to differ ($p \leq .10$).

[xyz] Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-12. Normalized dorsal by palmar RBAE in mm Al equivalence (mean ± SE).

day	0	32	50	64	82	96	128
Site 1							
Con	0.000 ± 0.011 [xy]	0.023 ± 0.011 [xz]	0.015 ± 0.011 [xyz]	-0.003 ± 0.011 [a, y]	0.017 ± 0.011 [xyz]	0.009 ± 0.011 [b, xyz]	0.025 ± 0.011 [z]
eST	0.000 ± 0.010 [y]	0.019 ± 0.010 [yz]	0.020 ± 0.011 [yz]	0.026 ± 0.011 [z]	0.034 ± 0.011 [z]	0.034 ± 0.010 [z]	0.039 ± 0.010 [z]
Site 2							
Con	0.000 ± 0.012 [x]	0.007 ± 0.013 [xy]	0.026 ± 0.012 [y]	-0.006 ± 0.012 [a, x]	0.019 ± 0.012 [xy]	0.008 ± 0.012 [xy]	0.015 ± 0.012 [b, xy]
eST	0.000 ± 0.012 [x]	0.022 ± 0.012 [xy]	0.035 ± 0.013 [yz]	0.029 ± 0.012 [yz]	0.036 ± 0.012 [yz]	0.034 ± 0.012 [yz]	0.047 ± 0.012 [z]
Site 3							
Con	0.000 ± 0.013 [z]	0.009 ± 0.013 [z]	0.026 ± 0.013 [z]	0.010 ± 0.013 [z]	0.025 ± 0.013 [z]	0.005 ± 0.013 [z]	0.021 ± 0.013 [z]
eST	0.000 ± 0.013 [x]	0.018 ± 0.013 [xy]	0.048 ± 0.014 [z]	0.030 ± 0.013 [yz]	0.031 ± 0.014 [yz]	0.032 ± 0.013 [yz]	0.052 ± 0.013 [z]
Site 4							
Con	0.000 ± 0.015 [z]	0.001 ± 0.016 [z]	-0.000 ± 0.016 [z]	-0.005 ± 0.015 [z]	0.011 ± 0.015 [z]	-0.015 ± 0.015 [a, z]	0.013 ± 0.015 [b, z]
eST	0.000 ± 0.014 [y]	0.020 ± 0.014 [y]	0.027 ± 0.016 [yz]	0.026 ± 0.014 [yz]	0.031 ± 0.016 [yz]	0.028 ± 0.014 [yz]	0.050 ± 0.015 [z]

[a] Treatments differ ($p \leq .05$).

[b] Trend for treatments to differ ($p \leq .10$).

[xyz] Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-13. Lateral by palmar RBAE in mm Al equivalence (mean ± SE).

day	0	32	50	64	82	96	128
Site 1							
Con	1.22 ± 0.02 ^{x}	1.31 ± 0.03 ^{yz}	1.24 ± 0.03 ^{x}	1.25 ± 0.02 ^{x}	1.26 ± 0.03 ^{xy}	1.25 ± 0.02 ^{xy}	1.32 ± 0.02 ^{z}
eST	1.22 ± 0.02 ^{x}	1.26 ± 0.02 ^{xyz}	1.26 ± 0.02 ^{xy}	1.25 ± 0.02 ^{xy}	1.25 ± 0.02 ^{xy}	1.28 ± 0.02 ^{yz}	1.31 ± 0.02 ^{z}
Site 2							
Con	1.31 ± 0.03 ^{x}	1.39 ± 0.03 ^{yz}	1.33 ± 0.03 ^{xy}	1.35 ± 0.03 ^{xy}	1.35 ± 0.03 ^{xy}	1.35 ± 0.03 ^{xy}	1.43 ± 0.03 ^{z}
eST	1.29 ± 0.03 ^{x}	1.36 ± 0.03 ^{y}	1.36 ± 0.03 ^{xy}	1.35 ± 0.03 ^{xy}	1.35 ± 0.03 ^{xy}	1.37 ± 0.03 ^{yz}	1.44 ± 0.03 ^{z}
Site 3							
Con	1.33 ± 0.03 ^{x}	1.42 ± 0.03 ^{yz}	1.34 ± 0.03 ^{x}	1.38 ± 0.03 ^{xy}	1.37 ± 0.03 ^{xy}	1.37 ± 0.03 ^{xy}	1.48 ± 0.03 ^{z}
eST	1.31 ± 0.03 ^{x}	1.39 ± 0.03 ^{y}	1.38 ± 0.04 ^{xy}	1.36 ± 0.03 ^{xy}	1.37 ± 0.03 ^{xy}	1.40 ± 0.03 ^{y}	1.48 ± 0.03 ^{z}
Site 4							
Con	1.25 ± 0.03 ^{x}	1.34 ± 0.03 ^{yz}	1.26 ± 0.04 ^{xy}	1.27 ± 0.03 ^{xy}	1.27 ± 0.03 ^{xy}	1.28 ± 0.03 ^{xy}	1.40 ± 0.03 ^{z}
eST	1.22 ± 0.03 ^{x}	1.31 ± 0.03 ^{y}	1.28 ± 0.04 ^{xy}	1.31 ± 0.03 ^{y}	1.28 ± 0.03 ^{xy}	1.33 ± 0.03 ^{yz}	1.40 ± 0.03 ^{z}

^{xyz} Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-14. Normalized lateral by palmar RBAE in mm Al equivalence (mean ± SE).

day	0	32	50	64	82	96	128
Site 1							
Con	0.000 ± 0.026 ^{x}	0.082 ± 0.028 ^{yz}	0.020 ± 0.027 ^{x}	0.026 ± 0.026 ^{y}	0.036 ± 0.027 ^{xy}	0.028 ± 0.026 ^{xy}	0.097 ± 0.026 ^{z}
eST	0.000 ± 0.025 ^{x}	0.045 ± 0.025 ^{xyz}	0.036 ± 0.027 ^{xy}	0.033 ± 0.025 ^{xy}	0.033 ± 0.027 ^{xy}	0.063 ± 0.025 ^{yz}	0.092 ± 0.025 ^{z}
Site 2							
Con	0.000 ± 0.032 ^{x}	0.084 ± 0.034 ^{yz}	0.022 ± 0.033 ^{xy}	0.043 ± 0.032 ^{xy}	0.047 ± 0.033 ^{xy}	0.043 ± 0.032 ^{xy}	0.123 ± 0.032 ^{z}
eST	0.000 ± 0.031 ^{x}	0.073 ± 0.031 ^{y}	0.066 ± 0.034 ^{xy}	0.057 ± 0.032 ^{xy}	0.062 ± 0.032 ^{xy}	0.084 ± 0.031 ^{y}	0.147 ± 0.031 ^{z}
Site 3							
Con	0.000 ± 0.035 ^{x}	0.090 ± 0.037 ^{yz}	0.013 ± 0.037 ^{xy}	0.049 ± 0.036 ^{xy}	0.043 ± 0.036 ^{xy}	0.044 ± 0.035 ^{xy}	0.146 ± 0.035 ^{z}
eST	0.000 ± 0.035 ^{x}	0.075 ± 0.035 ^{y}	0.066 ± 0.039 ^{xy}	0.052 ± 0.035 ^{xy}	0.071 ± 0.038 ^{xy}	0.083 ± 0.035 ^{y}	0.166 ± 0.036 ^{z}
Site 4							
Con	0.000 ± 0.037 ^{x}	0.087 ± 0.040 ^{yz}	0.019 ± 0.041 ^{xy}	0.030 ± 0.038 ^{xy}	0.035 ± 0.038 ^{xy}	0.030 ± 0.037 ^{xy}	0.156 ± 0.038 ^{z}
eST	0.000 ± 0.036 ^{x}	0.082 ± 0.036 ^{y}	0.055 ± 0.041 ^{xy}	0.083 ± 0.036 ^{y}	0.063 ± 0.039 ^{xy}	0.098 ± 0.036 ^{yz}	0.170 ± 0.037 ^{z}

^{xyz} Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-15. Medial by lateral RBAE in mm Al equivalence (mean ± SE).

day	0	32	50	64	82	96	128
Site 1							
Con	1.07 ± 0.02 ^{y}	1.07 ± 0.02 ^{y}	1.10 ± 0.02 ^{yz}	1.10 ± 0.02 ^{yz}	1.12 ± 0.02 ^{z}	1.10 ± 0.02 ^{yz}	1.08 ± 0.02 ^{yz}
eST	1.05 ± 0.02 ^{y}	1.09 ± 0.02 ^{y}	1.11 ± 0.02 ^{yz}	1.11 ± 0.02 ^{yz}	1.12 ± 0.02 ^{z}	1.10 ± 0.02 ^{yz}	1.10 ± 0.02 ^{yz}
Site 2							
Con	1.08 ± 0.02 ^{z}	1.08 ± 0.02 ^{z}	1.13 ± 0.02 ^{z}	1.11 ± 0.02 ^{z}	1.13 ± 0.02 ^{z}	1.11 ± 0.02 ^{z}	1.08 ± 0.02 ^{z}
eST	1.07 ± 0.02 ^{y}	1.09 ± 0.02 ^{yz}	1.13 ± 0.02 ^{z}	1.12 ± 0.02 ^{z}	1.13 ± 0.02 ^{z}	1.11 ± 0.02 ^{yz}	1.10 ± 0.02 ^{yz}
Site 3							
Con	1.09 ± 0.02 ^{xy}	1.09 ± 0.02 ^{xyz}	1.14 ± 0.02 ^{z}	1.11 ± 0.02 ^{xyz}	1.14 ± 0.02 ^{xz}	1.12 ± 0.02 ^{xyz}	1.08 ± 0.02 ^{y}
eST	1.09 ± 0.02 ^{y}	1.10 ± 0.02 ^{yz}	1.15 ± 0.02 ^{z}	1.15 ± 0.02 ^{z}	1.12 ± 0.02 ^{yz}	1.11 ± 0.02 ^{yz}	1.00 ± 0.02 ^{yz}
Site 4							
Con	1.10 ± 0.02 ^{yz}	1.11 ± 0.02 ^{yz}	1.15 ± 0.02 ^{y}	1.12 ± 0.02 ^{yz}	1.14 ± 0.02 ^{y}	1.12 ± 0.02 ^{yz}	1.08 ± 0.02 ^{z}
eST	1.11 ± 0.02 ^{z}	1.12 ± 0.02 ^{z}	1.15 ± 0.02 ^{z}	1.13 ± 0.02 ^{z}	1.15 ± 0.02 ^{z}	1.11 ± 0.02 ^{z}	1.10 ± 0.02 ^{z}

^{xyz} Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-16. Normalized medial by lateral RBAE in mm Al equivalence (mean ± SE).

day	0	32	50	64	82	96	128
Site 1							
Con	0.000 ± 0.017 [y]	-0.001 ± 0.017 [y]	0.026 ± 0.017 [yz]	0.023 ± 0.017 [yz]	0.044 ± 0.017 [z]	0.028 ± 0.017 [yz]	0.008 ± 0.017 [a, yz]
eST	0.000 ± 0.016 [x]	0.032 ± 0.016 [xy]	0.059 ± 0.017 [yz]	0.057 ± 0.016 [yz]	0.072 ± 0.017 [z]	0.050 ± 0.016 [yz]	0.049 ± 0.016 [yz]
Site 2							
Con	0.000 ± 0.023 [z]	0.001 ± 0.024 [z]	0.046 ± 0.024 [z]	0.024 ± 0.023 [z]	0.045 ± 0.024 [z]	0.0298 ± 0.023 [z]	0.001 ± 0.023 [z]
eST	0.000 ± 0.022 [y]	0.018 ± 0.022 [yz]	0.057 ± 0.023 [z]	0.048 ± 0.022 [z]	0.053 ± 0.023 [z]	0.039 ± 0.022 [yz]	0.024 ± 0.022 [yz]
Site 3							
Con	0.000 ± 0.026 [y]	0.005 ± 0.027 [yz]	0.059 ± 0.027 [z]	0.027 ± 0.026 [yz]	0.056 ± 0.027 [z]	0.033 ± 0.026 [yz]	-0.004 ± 0.026 [y]
eST	0.000 ± 0.025 [y]	0.013 ± 0.025 [yz]	0.058 ± 0.026 [z]	0.058 ± 0.025 [z]	0.037 ± 0.026 [yz]	0.026 ± 0.025 [yz]	0.011 ± 0.025 [yz]
Site 4							
Con	0.000 ± 0.031 [yz]	0.003 ± 0.031 [yz]	0.047 ± 0.031 [y]	0.021 ± 0.031 [yz]	0.044 ± 0.031 [y]	0.016 ± 0.031 [yz]	-0.021 ± 0.031 [z]
eST	0.000 ± 0.030 [z]	0.008 ± 0.030 [z]	0.044 ± 0.031 [z]	0.025 ± 0.030 [z]	0.048 ± 0.031 [z]	-0.002 ± 0.030 [z]	-0.010 ± 0.030 [z]

[a] Trend for treatments to differ ($p \leq .10$).

[xyz] Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-17. Medial by palmar RBAE in mm Al equivalence (mean ± SE).

day	0	32	50	64	82	96	128
Site 1							
Con	1.31 ± 0.03 ^{x}	1.39 ± 0.03 ^{yz}	1.36 ± 0.03 ^{y}	1.36 ± 0.03 ^{y}	1.40 ± 0.03 ^{yz}	1.37 ± 0.03 ^{yz}	1.42 ± 0.03 ^{z}
eST	1.28 ± 0.02 ^{x}	1.37 ± 0.02 ^{y}	1.39 ± 0.03 ^{yz}	1.39 ± 0.02 ^{y}	1.40 ± 0.03 ^{yz}	1.41 ± 0.02 ^{yz}	1.44 ± 0.02 ^{z}
Site 2							
Con	1.41 ± 0.03 ^{y}	1.50 ± 0.03 ^{z}	1.49 ± 0.03 ^{z}	1.49 ± 0.03 ^{z}	1.52 ± 0.03 ^{z}	1.49 ± 0.03 ^{z}	1.54 ± 0.03 ^{z}
eST	1.38 ± 0.03 ^{x}	1.49 ± 0.03 ^{y}	1.53 ± 0.03 ^{yz}	1.51 ± 0.03 ^{y}	1.52 ± 0.03 ^{yz}	1.52 ± 0.03 ^{yz}	1.57 ± 0.03 ^{z}
Site 3							
Con	1.44 ± 0.03 ^{y}	1.54 ± 0.03 ^{z}	1.54 ± 0.03 ^{z}	1.53 ± 0.03 ^{z}	1.56 ± 0.03 ^{z}	1.53 ± 0.03 ^{z}	1.59 ± 0.03 ^{z}
eST	1.43 ± 0.03 ^{x}	1.53 ± 0.03 ^{y}	1.57 ± 0.03 ^{yz}	1.56 ± 0.03 ^{yz}	1.54 ± 0.03 ^{y}	1.55 ± 0.03 ^{y}	1.62 ± 0.03 ^{z}
Site 4							
Con	1.39 ± 0.03 ^{x}	1.47 ± 0.03 ^{yz}	1.46 ± 0.03 ^{xyz}	1.42 ± 0.03 ^{xy}	1.45 ± 0.03 ^{xyz}	1.42 ± 0.03 ^{xy}	1.50 ± 0.03 ^{z}
eST	1.36 ± 0.03 ^{x}	1.45 ± 0.03 ^{y}	1.47 ± 0.04 ^{yz}	1.48 ± 0.03 ^{yz}	1.48 ± 0.03 ^{yz}	1.46 ± 0.03 ^{y}	1.53 ± 0.03 ^{z}

^{xyz} Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-18. Normalized medial by palmar RBAE in mm Al equivalence (mean ± SE).

day	0	32	50	64	82	96	128
Site 1							
Con	0.000 ± 0.028 ^{x}	0.086 ± 0.029 ^{yz}	0.057 ± 0.028 ^{y}	0.056 ± 0.028 ^{y}	0.096 ± 0.028 ^{yz}	0.065 ± 0.028 ^{yz}	0.115 ± 0.028 ^{z}
eST	0.000 ± 0.027 ^{x}	0.089 ± 0.027 ^{y}	0.108 ± 0.028 ^{yz}	0.105 ± 0.027 ^{y}	0.122 ± 0.028 ^{yz}	0.126 ± 0.027 ^{yz}	0.157 ± 0.027 ^{z}
Site 2							
Con	0.000 ± 0.031 ^{y}	0.088 ± 0.032 ^{z}	0.084 ± 0.031 ^{z}	0.075 ± 0.030 ^{z}	0.112 ± 0.031 ^{z}	0.082 ± 0.030 ^{z}	0.132 ± 0.030 ^{z}
eST	0.000 ± 0.029 ^{x}	0.107 ± 0.029 ^{y}	0.150 ± 0.032 ^{yz}	0.130 ± 0.030 ^{y}	0.139 ± 0.030 ^{yz}	0.143 ± 0.029 ^{yz}	0.192 ± 0.029 ^{z}
Site 3							
Con	0.000 ± 0.030 ^{y}	0.098 ± 0.032 ^{z}	0.096 ± 0.032 ^{z}	0.088 ± 0.031 ^{z}	0.122 ± 0.031 ^{z}	0.089 ± 0.030 ^{z}	0.149 ± 0.030 ^{z}
eST	0.000 ± 0.030 ^{x}	0.096 ± 0.030 ^{y}	0.134 ± 0.034 ^{yz}	0.127 ± 0.030 ^{yz}	0.113 ± 0.033 ^{yz}	0.116 ± 0.030 ^{yz}	0.176 ± 0.031 ^{z}
Site 4							
Con	0.000 ± 0.032 ^{x}	0.073 ± 0.034 ^{yz}	0.066 ± 0.035 ^{xyz}	0.038 ± 0.033 $^{a, xy}$	0.088 ± 0.033 ^{yz}	0.033 ± 0.032 $^{a, xy}$	0.114 ± 0.033 ^{z}
eST	0.000 ± 0.031 ^{y}	0.101 ± 0.031 ^{z}	0.112 ± 0.035 ^{z}	0.118 ± 0.031 ^{z}	0.122 ± 0.034 ^{z}	0.107 ± 0.031 ^{z}	0.166 ± 0.032 ^{z}

^{a} Trend for treatments to differ ($p \leq .10$).

^{xyz} Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-19. Dorsal cortical bone width micrometer readings in mm (mean ± SE).

day	0			64			128		
Site 1									
Con	11.9243	±	0.5031 [z]	12.3161	±	0.5031 [yz]	13.0104	±	0.5031 [y]
eST	11.4877	±	0.4860 [y]	11.9970	±	0.4860 [y]	13.9570	±	0.4860 [z]
Site 2									
Con	11.6093	±	0.4767 [y]	11.9771	±	0.4767 [yz]	12.7446	±	0.4767 [z]
eST	11.1283	±	0.4605 [y]	11.6677	±	0.4605 [y]	13.7017	±	0.4605 [z]
Site 3									
Con	10.9300	±	0.4314 [y]	11.3075	±	0.4314 [y]	12.1154	±	0.4314 [z]
eST	10.4557	±	0.4168 [y]	10.7980	±	0.4168 [y]	12.8560	±	0.4168 [z]
Site 4									
Con	7.2889	±	0.3218 [y]	7.5689	±	0.3218 [y]	8.6918	±	0.3218 [z]
eST	7.1160	±	0.3109 [y]	7.4473	±	0.3109 [y]	9.0100	±	0.3109 [z]

[yz] Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-20. Normalized dorsal cortical bone width micrometer readings in mm (mean ± SE).

day	0			64			128		
Site 1									
Con	0.0000	±	0.3188 [y]	0.3918	±	0.3188 [yz]	1.0861	±	0.3188 [a,z]
eST	0.0000	±	0.3080 [y]	0.5093	±	0.3080 [y]	2.4693	±	0.3080 [z]
Site 2									
Con	0.0000	±	0.3167 [y]	0.3679	±	0.3167 [yz]	1.1354	±	0.3167 [a,z]
eST	0.0000	±	0.3060 [y]	0.5393	±	0.3060 [y]	2.5733	±	0.3060 [z]
Site 3									
Con	0.0000	±	0.2930 [y]	0.3775	±	0.2930 [y]	1.1854	±	0.2930 [a,z]
eST	0.0000	±	0.2831 [y]	0.3423	±	0.2831 [y]	2.4003	±	0.2831 [z]
Site 4									
Con	0.0000	±	0.1987 [y]	0.2800	±	0.1987 [y]	1.4029	±	0.1987 [b,z]
eST	0.0000	±	0.1920 [y]	0.3313	±	0.1920 [y]	1.8940	±	0.1920 [z]

[a] Treatments differ ($p \leq .01$).

[b] Trend for treatments to differ ($p \leq .10$).

[yz] Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-21. Lateral cortical bone width micrometer readings in mm (mean ± SE).

day	0			64			128		
Site 1									
Con	10.5232	±	0.3417 [z]	10.7296	±	0.3417 [z]	10.5950	±	0.3417 [z]
eST	10.4437	±	0.3302 [z]	10.5367	±	0.3302 [z]	10.5313	±	0.3302 [z]
Site 2									
Con	10.7064	±	0.3696 [z]	10.8854	±	0.3696 [z]	10.7796	±	0.3696 [z]
eST	10.5857	±	0.3571 [z]	10.7647	±	0.3571 [z]	10.5767	±	0.3571 [z]
Site 3									
Con	10.3839	±	0.3677 [yz]	10.6179	±	0.3677 [y]	10.3232	±	0.3677 [z]
eST	10.2167	±	0.3552 [z]	10.4243	±	0.3552 [z]	10.3557	±	0.3552 [z]
Site 4									
Con	8.4393	±	0.3172 [z]	8.5750	±	0.3172 [z]	8.7346	±	0.3172 [z]
eST	7.9810	±	0.3065 [z]	8.1173	±	0.3065 [z]	8.0460	±	0.3065 [z]

[yz] Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-22. Normalized lateral cortical bone width micrometer readings in mm (mean ± SE).

day	0			64			128		
Site 1									
Con	0.0000	±	0.08141 [z]	0.2064	±	0.08141 [z]	0.0718	±	0.08141 [z]
eST	0.0000	±	0.07865 [z]	0.0930	±	0.07865 [z]	0.0877	±	0.07865 [z]
Site 2									
Con	0.0000	±	0.08811 [z]	0.1789	±	0.08811 [z]	0.0732	±	0.08811 [z]
eST	0.0000	±	0.08512 [z]	0.1790	±	0.08512 [z]	-0.0090	±	0.08512 [z]
Site 3									
Con	0.0000	±	0.1138 [yz]	0.2339	±	0.1138 [y]	-0.0607	±	0.1138 [z]
eST	0.0000	±	0.1099 [z]	0.2077	±	0.1099 [z]	0.1390	±	0.1099 [z]
Site 4									
Con	0.0000	±	0.1731 [z]	0.1357	±	0.1731 [z]	0.2954	±	0.1731 [z]
eST	0.0000	±	0.1673 [z]	0.1363	±	0.1673 [z]	0.0650	±	0.1673 [z]

[yz] Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-23. Medial cortical bone width micrometer readings in mm (mean ± SE).

day	0			64			128		
Site 1									
Con	13.0911	±	0.4780 [z]	13.2061	±	0.4780 [z]	13.3004	±	0.4780 [z]
eST	12.4007	±	0.4618 [y]	12.3930	±	0.4618 [y]	13.1040	±	0.4618 [z]
Site 2									
Con	12.8529	±	0.5165 [y]	13.0118	±	0.5165 [yz]	13.2993	±	0.5165 [z]
eST	12.1653	±	0.4990 [y]	12.2473	±	0.4990 [y]	12.7957	±	0.4990 [z]
Site 3									
Con	12.8289	±	0.5133 [z]	13.0661	±	0.5133 [z]	13.1404	±	0.5133 [z]
eST	12.1660	±	0.4959 [y]	12.1557	±	0.4959 [y]	12.7267	±	0.4959 [z]
Site 4									
Con	11.2800	±	0.4904 [z]	11.4693	±	0.4904 [z]	11.2279	±	0.4904 [z]
eST	10.3957	±	0.4737 [y]	10.7853	±	0.4737 [yz]	11.2057	±	0.4737 [z]

[yz] Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-24. Normalized medial cortical bone width micrometer readings in mm (mean ± SE).

day	0			64			128		
Site 1									
Con	0.0000	±	0.2103 [z]	0.1150	±	0.2103 [z]	0.2093	±	0.2103 [a,z]
eST	0.0000	±	0.2032 [y]	-0.0013	±	0.2032 [y]	0.7033	±	0.2032 [z]
Site 2									
Con	0.0000	±	0.1785 [y]	0.1589	±	0.1785 [yz]	0.4464	±	0.1785 [z]
eST	0.0000	±	0.1724 [y]	0.0820	±	0.1724 [y]	0.6303	±	0.1724 [z]
Site 3									
Con	0.0000	±	0.1822 [z]	0.2371	±	0.1822 [z]	0.3114	±	0.1822 [z]
eST	0.0000	±	0.1760 [y]	-0.0103	±	0.1760 [y]	0.5607	±	0.1760 [z]
Site 4									
Con	0.0000	±	0.2678 [z]	0.1893	±	0.2678 [z]	-0.0521	±	0.2678 [b,z]
eST	0.0000	±	0.2587 [y]	0.3897	±	0.2587 [yz]	0.8100	±	0.2587 [z]

[a] Trend for treatments to differ ($p \leq .10$).

[b] Treatments differ ($p \leq .05$).

[yz] Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-25. Palmar cortical bone width micrometer readings in mm (mean ± SE).

day	0			64			128		
Site 1									
Con	6.7646	±	0.3475 [z]	6.6279	±	0.3475 [z]	6.5589	±	0.3475 [z]
eST	6.6000	±	0.3357 [z]	6.8100	±	0.3357 [z]	6.8260	±	0.3357 [z]
Site 2									
Con	6.8139	±	0.2547 [z]	6.7189	±	0.2547 [z]	6.7800	±	0.2547 [z]
eST	6.8713	±	0.2461 [z]	7.0480	±	0.2461 [z]	6.9557	±	0.2461 [z]
Site 3									
Con	6.7993	±	0.2582 [z]	6.6464	±	0.2582 [z]	6.7875	±	0.2582 [z]
eST	6.6790	±	0.2495 [z]	6.9177	±	0.2495 [z]	6.9900	±	0.2495 [z]
Site 4									
Con	4.9689	±	0.2370 [y]	5.0461	±	0.2370 [yz]	5.4661	±	0.2370 [z]
eST	4.9350	±	0.2289 [y]	5.3300	±	0.2289 [y]	5.8183	±	0.2289 [z]

[yz] Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-26. Normalized palmar cortical bone width micrometer readings in mm (mean ± SE).

day	0			64			128		
Site 1									
Con	0.0000	±	0.2737 [z]	-0.1368	±	0.2737 [z]	-0.2057	±	0.2737 [z]
eST	0.0000	±	0.2644 [z]	0.2100	±	0.2644 [z]	0.2260	±	0.2644 [z]
Site 2									
Con	0.0000	±	0.1660 [z]	-0.0950	±	0.1660 [z]	-0.0339	±	0.1660 [z]
eST	0.0000	±	0.1604 [z]	0.1767	±	0.1604 [z]	0.0843	±	0.1604 [z]
Site 3									
Con	0.0000	±	0.1962 [z]	-0.1529	±	0.1962 [z]	-0.0118	±	0.1962 [z]
eST	0.0000	±	0.1895 [z]	0.2387	±	0.1895 [z]	0.3110	±	0.1895 [z]
Site 4									
Con	0.0000	±	0.1903 [y]	0.0771	±	0.1903 [yz]	0.4971	±	0.1903 [z]
eST	0.0000	±	0.1838 [y]	0.3950	±	0.1838 [y]	0.8833	±	0.1838 [z]

[yz] Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-27. Dorsal to palmar medullary cavity width micrometer readings in mm (mean ± SE).

day	0			64			128		
Site 1									
Con	12.3736	±	0.4408 [z]	12.2661	±	0.4408 [z]	12.2036	±	0.4408 [z]
eST	12.2757	±	0.4258 [y]	11.9853	±	0.4258 [y]	11.6130	±	0.4258 [z]
Site 2									
Con	13.1675	±	0.4298 [z]	13.1564	±	0.4298 [z]	12.9518	±	0.4298 [z]
eST	13.0883	±	0.4152 [y]	12.6663	±	0.4152 [z]	12.4273	±	0.4152 [z]
Site 3									
Con	13.7611	±	0.4265 [z]	13.7586	±	0.4265 [z]	13.7854	±	0.4265 [z]
eST	13.5443	±	0.4121 [y]	13.3757	±	0.4121 [yz]	13.1643	±	0.4121 [z]
Site 4									
Con	19.2679	±	0.5088 [z]	19.2793	±	0.5088 [z]	19.1836	±	0.5088 [z]
eST	18.9760	±	0.4915 [z]	18.8953	±	0.4915 [z]	18.5660	±	0.4915 [z]

[yz] Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-28. Normalized dorsal to palmar medullary cavity width micrometer readings in mm (mean ± SE).

day	0			64			128		
Site 1									
Con	0.0000	±	0.1519 [z]	-0.1075	±	0.1519 [z]	-0.1700	±	0.1519 [a,z]
eST	0.0000	±	0.1468 [y]	-0.2903	±	0.1468 [y]	-0.6627	±	0.1468 [z]
Site 2									
Con	0.0000	±	0.1233 [z]	-0.0111	±	0.1233 [a,z]	-0.2157	±	0.1233 [a,z]
eST	0.0000	±	0.1192 [y]	-0.4220	±	0.1192 [z]	-0.6610	±	0.1192 [z]
Site 3									
Con	0.0000	±	0.1261 [z]	-0.0025	±	0.1261 [z]	0.0243	±	0.1261 [a,z]
eST	0.0000	±	0.1219 [y]	-0.1687	±	0.1219 [yz]	-0.3800	±	0.1219 [z]
Site 4									
Con	0.0000	±	0.1725 [z]	0.0114	±	0.1725 [z]	-0.0843	±	0.1725 [z]
eST	0.0000	±	0.1667 [z]	-0.0807	±	0.1667 [z]	-0.4100	±	0.1667 [z]

[a] Treatments differ ($p \leq .05$).

Table A-29. Dorsal to palmar bone diameter micrometer readings in mm (mean ± SE).

day	0			64			128		
Site 1									
Con	31.0625	±	0.6095 [z]	31.2100	±	0.6095 [z]	31.7729	±	0.6095 [z]
eST	30.3633	±	0.5888 [y]	30.7923	±	0.5888 [y]	32.3960	±	0.5888 [z]
Site 2									
Con	31.5907	±	0.5450 [z]	31.8525	±	0.5450 [z]	32.4764	±	0.5450 [z]
eST	31.0880	±	0.5265 [y]	31.3820	±	0.5265 [y]	33.0847	±	0.5265 [z]
Site 3									
Con	31.4904	±	0.5379 [y]	31.7125	±	0.5379 [y]	32.6882	±	0.5379 [z]
eST	30.6790	±	0.5197 [y]	31.0913	±	0.5197 [y]	33.0103	±	0.5197 [z]
Site 4									
Con	31.5257	±	0.5447 [y]	31.8943	±	0.5447 [y]	33.3414	±	0.5447 [z]
eST	31.0270	±	0.5262 [y]	31.6727	±	0.5262 [y]	33.3943	±	0.5262 [z]

[yz] Means in the same row not sharing the same superscript differ (p≤.05).

Table A-30. Normalized dorsal to palmar bone diameter micrometer readings in mm (mean ± SE).

day	0			64			128		
Site 1									
Con	0.0000	±	0.4835 [z]	0.1475	±	0.4835 [z]	0.7104	±	0.4835 [a,z]
eST	0.0000	±	0.4671 [y]	0.4290	±	0.4671 [y]	2.0327	±	0.4671 [z]
Site 2									
Con	0.0000	±	0.3845 [z]	0.2618	±	0.3845 [z]	0.8857	±	0.3845 [b,z]
eST	0.0000	±	0.3714 [y]	0.2940	±	0.3714 [y]	1.9967	±	0.3714 [z]
Site 3									
Con	0.0000	±	0.3729 [y]	0.2221	±	0.3729 [y]	1.1979	±	0.3729 [a,z]
eST	0.0000	±	0.3602 [y]	0.4123	±	0.3602 [y]	2.3313	±	0.3602 [z]
Site 4									
Con	0.0000	±	0.3144 [y]	0.3686	±	0.3144 [y]	1.8157	±	0.3144 [z]
eST	0.0000	±	0.3038 [y]	0.6457	±	0.3038 [y]	2.3673	±	0.3038 [z]

[a] Treatments differ (p≤.05).

[b] Treatments differ (p≤.05).

[yz] Means in the same row not sharing the same superscript differ (p≤.05).

Table A-31. Lateral to medial medullary cavity width micrometer readings in mm (mean ± SE).

day	0	64	128
Site 1			
Con	17.5514 ± 0.7477 [z]	17.6093 ± 0.7477 [z]	17.3850 ± 0.7477 [z]
eST	17.1853 ± 0.7223 [z]	16.9907 ± 0.7223 [z]	17.1130 ± 0.7223 [z]
Site 2			
Con	16.9950 ± 0.7181 [z]	17.0496 ± 0.7181 [z]	16.9889 ± 0.7181 [z]
eST	16.9397 ± 0.6937 [z]	16.7453 ± 0.6937 [z]	16.8397 ± 0.6937 [z]
Site 3			
Con	17.4179 ± 0.6850 [z]	17.3346 ± 0.6850 [z]	17.3989 ± 0.6850 [z]
eST	17.3380 ± 0.6618 [z]	17.2943 ± 0.6618 [z]	17.0303 ± 0.6618 [z]
Site 4			
Con	23.9496 ± 0.7655 [z]	23.9979 ± 0.7655 [z]	23.9886 ± 0.7655 [z]
eST	24.5763 ± 0.7395 [y]	23.8210 ± 0.7395 [z]	24.3103 ± 0.7395 [yz]

[yz] Means in the same row not sharing the same superscript differ (p≤.05).

Table A-32. Normalized lateral to medial medullary cavity width micrometer readings in mm (mean ± SE).

day	0	64	128
Site 1			
Con	0.0000 ± 0.1285 [z]	0.0579 ± 0.1285 [z]	-0.1664 ± 0.1285 [z]
eST	0.0000 ± 0.1241 [z]	-0.1947 ± 0.1241 [z]	-0.0723 ± 0.1241 [z]
Site 2			
Con	0.0000 ± 0.1189 [z]	0.0546 ± 0.1189 [z]	-0.0061 ± 0.1189 [z]
eST	0.0000 ± 0.1149 [z]	-0.1943 ± 0.1149 [z]	-0.1000 ± 0.1149 [z]
Site 3			
Con	0.0000 ± 0.1488 [z]	-0.0832 ± 0.1488 [z]	-0.0189 ± 0.1488 [z]
eST	0.0000 ± 0.1438 [z]	-0.0437 ± 0.1438 [z]	-0.3077 ± 0.1438 [z]
Site 4			
Con	0.0000 ± 0.2728 [z]	0.0482 ± 0.2728 [a,z]	0.0389 ± 0.2728 [z]
eST	0.0000 ± 0.2635 [y]	-0.7553 ± 0.2635 [z]	-0.2660 ± 0.2635 [yz]

[a] Treatments differ (p≤.05).

[yz] Means in the same row not sharing the same superscript differ (p≤.05).

Table A-33. Lateral to medial bone diameter micrometer readings in mm (mean ± SE).

day	0			64			128		
Site 1									
Con	41.1657	±	0.6575 [z]	41.5450	±	0.6575 [a, z]	41.2804	±	0.6575 [z]
eST	40.0297	±	0.6352 [y]	39.9267	±	0.6352 [y]	40.7483	±	0.6352 [z]
Site 2									
Con	40.5543	±	0.6327 [z]	40.9468	±	0.6327 [z]	41.0679	±	0.6327 [z]
eST	39.6907	±	0.6112 [z]	39.7573	±	0.6112 [z]	40.2120	±	0.6112 [z]
Site 3									
Con	40.6307	±	0.6662 [z]	41.0186	±	0.6662 [z]	40.8625	±	0.6662 [z]
eST	39.7207	±	0.6436 [z]	39.8743	±	0.6436 [z]	40.1127	±	0.6436 [z]
Site 4									
Con	43.6689	±	0.7174 [z]	44.0421	±	0.7174 [z]	43.9511	±	0.7174 [z]
eST	42.9530	±	0.6931 [yz]	42.7237	±	0.6931 [y]	43.5620	±	0.6931 [z]

[a] Trend for treatments to differ ($p \leq .10$).

[yz] Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-34. Normalized lateral to medial bone diameter micrometer readings in mm (mean ± SE).

day	0			64			128		
Site 1									
Con	0.0000	±	0.2857 [z]	0.3793	±	0.2857 [z]	0.1146	±	0.2857 [z]
eST	0.0000	±	0.2760 [y]	-0.1030	±	0.2760 [y]	0.7187	±	0.2760 [z]
Site 2									
Con	0.0000	±	0.2367 [z]	0.3925	±	0.2367 [z]	0.5136	±	0.2367 [z]
eST	0.0000	±	0.2287 [z]	0.0667	±	0.2287 [z]	0.5213	±	0.2287 [z]
Site 3									
Con	0.0000	±	0.2432 [z]	0.3879	±	0.2432 [z]	0.2318	±	0.2432 [z]
eST	0.0000	±	0.2349 [z]	0.1537	±	0.2349 [z]	0.3920	±	0.2349 [z]
Site 4									
Con	0.0000	±	0.3127 [z]	0.3732	±	0.3127 [z]	0.2821	±	0.3127 [z]
eST	0.0000	±	0.3021 [yz]	-0.2293	±	0.3021 [y]	0.6090	±	0.3021 [z]

[yz] Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-35. Computed bone index value of micrometer readings at site 3, (mean ± SE).

day	0			64			128		
Site 3									
Con	2.2946	±	0.1996 [y]	2.3806	±	0.1996 [yz]	2.6156	±	0.19967 [z]
eST	2.2966	±	0.2760 [y]	2.4006	±	0.2760 [y]	3.0676	±	0.2760 [z]

[yz] Means in the same row not sharing the same superscript differ ($p \leq .05$).

Table A-36. Normalized computed bone index value of micrometer readings at site 3, (mean ± SE).

day	0			64			128		
Site 3									
Con	0.0000	±	0.1175 [y]	0.0860	±	0.1175 [yz]	0.3210	±	0.1175 [a,z]
eST	0.0000	±	0.1135 [y]	0.1039	±	0.1135 [y]	0.7710	±	0.1135 [z]

[a] Treatments differ ($p \leq .01$).

[yz] Means in the same row not sharing the same superscript differ ($p \leq .05$).

APPENDIX B

ANOVA TABLES

Table B-1. ANOVA table for total radiographic bone aluminum equivalency (mm^2Al) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	9.88	<.0001
Treatment	1	27.1	0.66	0.4223
Treatment*Day	6	155	1.22	0.3007

Table B-2. ANOVA table for normalized total radiographic bone aluminum equivalency (mm^2Al) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	9.86	<.0001
Treatment	1	27.1	2.08	0.1606
Treatment*Day	6	155	1.21	0.3064

Table B-3. ANOVA table for total radiographic bone aluminum equivalency (mm^2Al) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	11.66	<.0001
Treatment	1	27.1	0.57	0.4550
Treatment*Day	6	155	1.09	0.3736

Table B-4. ANOVA table for normalized total radiographic bone aluminum equivalency (mm^2Al) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	11.13	<.0001
Treatment	1	27.1	1.18	0.1892
Treatment*Day	6	155	1.05	0.3972

Table B-5. ANOVA table for total radiographic bone aluminum equivalency (mm^2Al) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	13.18	<.0001
Treatment	1	27.1	0.57	0.4567
Treatment*Day	6	155	1.04	0.3998

Table B-6. ANOVA table for normalized total radiographic bone aluminum equivalency (mm^2Al) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	13.15	<.0001
Treatment	1	27.1	1.85	0.1848
Treatment*Day	6	155	1.04	0.4015

Table B-7. ANOVA table for total radiographic bone aluminum equivalency (mm^2Al) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	12.82	<.0001
Treatment	1	27.1	0.22	0.6399
Treatment*Day	6	155	1.00	0.4244

Table B-8. ANOVA table for normalized total radiographic bone aluminum equivalency (mm^2Al) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	12.81	<.0001
Treatment	1	27.1	1.88	0.1811
Treatment*Day	6	155	1.01	0.4231

Table B-9. ANOVA table for dorsal radiographic bone aluminum equivalency (mm Al) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	154	12.09	<.0001
Treatment	1	27.1	3.14	0.0877
Treatment*Day	6	154	0.75	0.6119

Table B-10. ANOVA table for normalized dorsal radiographic bone aluminum equivalency (mm Al) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	12.12	<.0001
Treatment	1	27.3	0.56	0.4606
Treatment*Day	6	155	0.75	0.6135

Table B-11. ANOVA table for dorsal radiographic bone aluminum equivalency (mm Al) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	154	10.52	<.0001
Treatment	1	27.1	2.72	0.1103
Treatment*Day	6	154	0.62	0.7143

Table B-12. ANOVA table for normalized dorsal radiographic bone aluminum equivalency (mm Al) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	10.53	<.0001
Treatment	1	27.1	0.75	0.3932
Treatment*Day	6	155	0.62	0.7173

Table B-13. ANOVA table for dorsal radiographic bone aluminum equivalency (mm Al) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	154	9.41	<.0001
Treatment	1	27.1	2.93	0.0983
Treatment*Day	6	154	0.30	0.9371

Table B-14. ANOVA table for normalized dorsal radiographic bone aluminum equivalency (mm Al) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	9.11	<.0001
Treatment	1	27.1	0.95	0.3393
Treatment*Day	6	155	0.35	0.9072

Table B-15. ANOVA table for dorsal radiographic bone aluminum equivalency (mm Al) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	153	7.11	<.0001
Treatment	1	27.1	0.43	0.5166
Treatment*Day	6	153	0.48	0.8250

Table B-16. ANOVA table for normalized dorsal radiographic bone aluminum equivalency (mm Al) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	7.12	<.0001
Treatment	1	27.1	1.89	0.1804
Treatment*Day	6	155	0.51	0.8000

Table B-17. ANOVA table for lateral radiographic bone aluminum equivalency (mm Al) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	8.22	<.0001
Treatment	1	27	0.64	0.4316
Treatment*Day	6	155	0.25	0.9595

Table B-18. ANOVA table for normalized lateral radiographic bone aluminum equivalency (mm Al) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	156	8.28	<.0001
Treatment	1	27.2	0.01	0.9315
Treatment*Day	6	156	0.23	0.9648

Table B-19. ANOVA table for lateral radiographic bone aluminum equivalency (mm Al) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	9.08	<.0001
Treatment	1	27	0.53	0.4714
Treatment*Day	6	155	0.21	0.9747

Table B-20. ANOVA table for normalized lateral radiographic bone aluminum equivalency (mm Al) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	156	9.17	<.0001
Treatment	1	27.2	0.09	0.7720
Treatment*Day	6	156	0.20	0.9750

Table B-21. ANOVA table for lateral radiographic bone aluminum equivalency (mm Al) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	9.71	<.0001
Treatment	1	27	0.51	0.4820
Treatment*Day	6	155	0.33	0.9193

Table B-22. ANOVA table for normalized lateral radiographic bone aluminum equivalency (mm Al) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	156	9.80	<.0001
Treatment	1	27.2	0.12	0.7316
Treatment*Day	6	156	0.34	0.9125

Table B-23. ANOVA table for lateral radiographic bone aluminum equivalency (mm Al) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	13.36	<.0001
Treatment	1	27.1	0.52	0.4790
Treatment*Day	6	155	0.44	0.8527

Table B-24. ANOVA table for normalized lateral radiographic bone aluminum equivalency (mm Al) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	156	14.14	<.0001
Treatment	1	27.3	0.40	0.5319
Treatment*Day	6	156	0.35	0.9107

Table B-25. ANOVA table for medial radiographic bone aluminum equivalency (mm Al) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	19.50	<.0001
Treatment	1	27.1	0.36	0.5549
Treatment*Day	6	155	0.58	0.7441

Table B-26. ANOVA table for normalized medial radiographic bone aluminum equivalency (mm Al) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	19.53	<.0001
Treatment	1	27.2	1.33	0.2587
Treatment*Day	6	155	0.59	0.7354

Table B-27. ANOVA table for medial radiographic bone aluminum equivalency (mm Al) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	20.03	<.0001
Treatment	1	27.1	0.40	0.5306
Treatment*Day	6	155	0.43	0.8568

Table B-28. ANOVA table for normalized medial radiographic bone aluminum equivalency (mm Al) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	20.10	<.0001
Treatment	1	27.1	1.07	0.3109
Treatment*Day	6	155	0.46	0.8338

Table B-29. ANOVA table for medial radiographic bone aluminum equivalency (mm Al) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	18.69	<.0001
Treatment	1	27.1	0.35	0.5570
Treatment*Day	6	155	0.29	0.9391

Table B-30. ANOVA table for normalized medial radiographic bone aluminum equivalency (mm Al) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	156	18.71	<.0001
Treatment	1	27.1	0.50	0.4871
Treatment*Day	6	156	0.30	0.9348

Table B-31. ANOVA table for medial radiographic bone aluminum equivalency (mm Al) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	16.82	<.0001
Treatment	1	27.1	0.21	0.6488
Treatment*Day	6	155	0.64	0.6955

Table B-32. ANOVA table for normalized medial radiographic bone aluminum equivalency (mm Al) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	17.13	<.0001
Treatment	1	27.1	0.72	0.4027
Treatment*Day	6	155	0.61	0.7207

Table B-33. ANOVA table for palmar radiographic bone aluminum equivalency (mm Al) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	154	3.64	0.0021
Treatment	1	27	1.19	0.2856
Treatment*Day	6	154	0.30	0.9353

Table B-34. ANOVA table for normalized palmar radiographic bone aluminum equivalency (mm Al) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	156	3.68	0.0019
Treatment	1	27.3	0.02	0.8756
Treatment*Day	6	156	0.31	0.9329

Table B-35. ANOVA table for palmar radiographic bone aluminum equivalency (mm Al) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	151	3.33	0.0042
Treatment	1	27.1	0.83	0.3689
Treatment*Day	6	151	0.19	0.9804

Table B-36. ANOVA table for normalized palmar radiographic bone aluminum equivalency (mm Al) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	152	3.52	0.0027
Treatment	1	27.3	0.17	0.6798
Treatment*Day	6	152	0.20	0.9763

Table B-37. ANOVA table for palmar radiographic bone aluminum equivalency (mm Al) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	146	2.84	<.0121
Treatment	1	27	0.47	0.4975
Treatment*Day	6	146	0.22	0.9701

Table B-38. ANOVA table for normalized palmar radiographic bone aluminum equivalency (mm Al) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	144	2.75	0.0145
Treatment	1	27.8	0.06	0.8009
Treatment*Day	6	144	0.25	0.9594

Table B-39. ANOVA table for palmar radiographic bone aluminum equivalency (mm Al) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	143	2.02	0.0664
Treatment	1	27	1.16	0.2918
Treatment*Day	6	143	0.42	0.8621

Table B-40. ANOVA table for normalized palmar radiographic bone aluminum equivalency (mm Al) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	138	1.86	0.0913
Treatment	1	27.9	0.08	0.7728
Treatment*Day	6	138	0.49	0.8118

Table B-41. ANOVA table for ratio of dorsal to palmar radiographic bone aluminum equivalency (mm Al) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	154	2.99	0.0086
Treatment	1	26.9	0.93	0.3445
Treatment*Day	6	154	1.11	0.3615

Table B-42. ANOVA table for normalized ratio of dorsal to palmar radiographic bone aluminum equivalency (mm Al) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	3.04	0.0077
Treatment	1	27.1	1.52	0.2278
Treatment*Day	6	155	1.11	0.3619

Table B-43. ANOVA table for ratio of dorsal to palmar radiographic bone aluminum equivalency (mm Al) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	151	3.08	0.0071
Treatment	1	27.2	0.51	0.4811
Treatment*Day	6	151	1.02	0.4145

Table B-44. ANOVA table for normalized ratio of dorsal to palmar radiographic bone aluminum equivalency (mm Al) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	151	3.27	0.0047
Treatment	1	26.5	2.48	0.1273
Treatment*Day	6	151	1.01	0.4206

Table B-45. ANOVA table for ratio of dorsal to palmar radiographic bone aluminum equivalency (mm Al) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	145	3.04	0.0036
Treatment	1	27	0.47	0.4997
Treatment*Day	6	145	0.68	0.6658

Table B-46. ANOVA table for normalized ratio of dorsal to palmar radiographic bone aluminum equivalency (mm Al) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	142	3.47	0.0032
Treatment	1	25.8	1.55	0.2245
Treatment*Day	6	142	0.66	0.6800

Table B-47. ANOVA table for ratio of dorsal to palmar radiographic bone aluminum equivalency (mm Al) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	142	1.97	0.0735
Treatment	1	27.1	0.40	0.5316
Treatment*Day	6	142	1.06	0.3870

Table B-48. ANOVA table for normalized ratio of dorsal to palmar radiographic bone aluminum equivalency (mm Al) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	135	1.76	0.1119
Treatment	1	25	2.50	0.1263
Treatment*Day	6	135	0.87	0.5181

Table B-49. ANOVA table for ratio of lateral to palmar radiographic bone aluminum equivalency (mm Al) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	4.71	0.0002
Treatment	1	27.3	0.01	0.9190
Treatment*Day	6	155	0.63	0.7078

Table B-50. ANOVA table for normalized ratio of lateral to palmar radiographic bone aluminum equivalency (mm Al) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	4.69	0.0002
Treatment	1	27.1	0.01	0.9427
Treatment*Day	6	155	0.60	0.7262

Table B-51. ANOVA table for ratio of lateral to palmar radiographic bone aluminum equivalency (mm Al) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	152	6.04	<.0001
Treatment	1	27.5	0.00	0.9770
Treatment*Day	6	152	0.36	0.9054

Table B-52. ANOVA table for normalized ratio of lateral to palmar radiographic bone aluminum equivalency (mm Al) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	152	6.02	<.0001
Treatment	1	27.2	0.31	0.5818
Treatment*Day	6	152	0.34	0.9174

Table B-53. ANOVA table for ratio of lateral to palmar radiographic bone aluminum equivalency (mm Al) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	146	6.78	<.0001
Treatment	1	27.2	0.00	0.9971
Treatment*Day	6	146	0.44	0.8529

Table B-54. ANOVA table for normalized ratio of lateral to palmar radiographic bone aluminum equivalency (mm Al) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	143	6.35	<.0001
Treatment	1	25.8	0.26	0.6150
Treatment*Day	6	143	0.35	0.9063

Table B-55. ANOVA table for ratio of lateral to palmar radiographic bone aluminum equivalency (mm Al) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	144	6.76	<.0001
Treatment	1	27.2	0.40	0.7688
Treatment*Day	6	144	1.06	0.7490

Table B-56. ANOVA table for normalized ratio of lateral to palmar radiographic bone aluminum equivalency (mm Al) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	135	6.08	<.0001
Treatment	1	25.2	0.56	0.4597
Treatment*Day	6	135	0.45	0.8469

Table B-57. ANOVA table for ratio of medial to lateral radiographic bone aluminum equivalency (mm Al) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	156	3.95	0.0011
Treatment	1	27.1	0.18	0.6731
Treatment*Day	6	156	0.52	0.7915

Table B-58. ANOVA table for normalized ratio of medial to lateral radiographic bone aluminum equivalency (mm Al) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	4.06	0.0008
Treatment	1	26.6	2.81	0.1055
Treatment*Day	6	155	0.53	0.7878

Table B-59. ANOVA table for ratio of medial to lateral radiographic bone aluminum equivalency (mm Al) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	156	2.53	0.0232
Treatment	1	27.2	0.11	0.7427
Treatment*Day	6	156	0.13	0.9916

Table B-60. ANOVA table for normalized ratio of medial to lateral radiographic bone aluminum equivalency (mm Al) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	2.67	0.0170
Treatment	1	27	0.34	0.5655
Treatment*Day	6	155	0.13	0.9923

Table B-61. ANOVA table for ratio of medial to lateral radiographic bone aluminum equivalency (mm Al) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	156	2.60	0.0198
Treatment	1	27.2	0.13	0.7182
Treatment*Day	6	156	0.32	0.9233

Table B-62. ANOVA table for normalized ratio of medial to lateral radiographic bone aluminum equivalency (mm Al) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	2.72	0.0152
Treatment	1	27.1	0.02	0.8769
Treatment*Day	6	155	0.33	0.9208

Table B-63. ANOVA table for ratio of medial to lateral radiographic bone aluminum equivalency (mm Al) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	156	2.12	0.0543
Treatment	1	27.2	0.21	0.7278
Treatment*Day	6	156	0.10	0.9962

Table B-64. ANOVA table for normalized ratio of medial to lateral radiographic bone aluminum equivalency (mm Al) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	2.27	0.0393
Treatment	1	27.2	0.00	0.9872
Treatment*Day	6	155	0.10	0.9961

Table B-65. ANOVA table for ratio of medial to palmar radiographic bone aluminum equivalency (mm Al) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	155	9.80	<.0001
Treatment	1	27.2	0.10	0.7526
Treatment*Day	6	155	0.81	0.5674

Table B-66. ANOVA table for normalized ratio of medial to palmar radiographic bone aluminum equivalency (mm Al) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	154	9.83	<.0001
Treatment	1	27.2	1.27	0.2700
Treatment*Day	6	154	0.80	0.5721

Table B-67. ANOVA table for ratio of medial to palmar radiographic bone aluminum equivalency (mm Al) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	152	11.59	<.0001
Treatment	1	27.5	0.12	0.7270
Treatment*Day	6	152	0.74	0.6192

Table B-68. ANOVA table for normalized ratio of medial to palmar radiographic bone aluminum equivalency (mm Al) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	151	11.66	<.0001
Treatment	1	26.9	1.60	0.2162
Treatment*Day	6	151	0.73	0.6273

Table B-69. ANOVA table for ratio of medial to palmar radiographic bone aluminum equivalency (mm Al) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	146	9.89	<.0001
Treatment	1	27.3	0.07	0.7979
Treatment*Day	6	146	0.47	0.8308

Table B-70. ANOVA table for normalized ratio of medial to palmar radiographic bone aluminum equivalency (mm Al) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	143	9.56	<.0001
Treatment	1	26.1	0.30	0.5885
Treatment*Day	6	143	0.37	0.8945

Table B-71. ANOVA table for ratio of medial to palmar radiographic bone aluminum equivalency (mm Al) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	143	6.16	<.0001
Treatment	1	27.1	0.22	0.6456
Treatment*Day	6	143	0.75	0.6133

Table B-72. ANOVA table for normalized ratio of medial to palmar radiographic bone aluminum equivalency (mm Al) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	6	135	6.07	<.0001
Treatment	1	25.4	1.93	0.1769
Treatment*Day	6	135	0.64	0.6958

Table B-73. ANOVA table for dorsal cortical bone width micrometer readings (in mm) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	21.97	<.0001
Treatment	1	27	0.01	0.9191
Treatment*Day	2	54	3.78	0.0290

Table B-74. ANOVA table for normalized dorsal cortical bone width micrometer readings (in mm) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	21.97	<.0001
Treatment	1	27	2.70	0.1122
Treatment*Day	2	54	3.78	0.0290

Table B-75. ANOVA table for dorsal cortical bone width micrometer readings (in mm) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	24.13	<.0001
Treatment	1	27	0.01	0.9244
Treatment*Day	2	54	3.98	0.0244

Table B-76. ANOVA table for normalized dorsal cortical bone width micrometer readings (in mm) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	24.13	<.0001
Treatment	1	27	3.18	0.0860
Treatment*Day	2	54	3.98	0.0244

Table B-77. ANOVA table for dorsal cortical bone width micrometer readings (in mm) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	25.90	<.0001
Treatment	1	27	0.02	0.8766
Treatment*Day	2	54	3.65	0.0327

Table B-78. ANOVA table for normalized dorsal cortical bone width micrometer readings (in mm) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	25.90	<.0001
Treatment	1	27	2.11	0.1580
Treatment*Day	2	54	3.65	0.0327

Table B-79. ANOVA table for dorsal cortical bone width micrometer readings (in mm) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	44.29	<.0001
Treatment	1	27	0.00	0.9841
Treatment*Day	2	54	1.05	0.3571

Table B-80. ANOVA table for normalized dorsal cortical bone width micrometer readings (in mm) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	44.29	<.0001
Treatment	1	27	1.09	0.3061
Treatment*Day	2	54	1.05	0.3571

Table B-81. ANOVA table for lateral cortical bone width micrometer readings (in mm) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	1.86	0.1654
Treatment	1	27	0.06	0.8120
Treatment*Day	2	54	0.41	0.6642

Table B-82. ANOVA table for normalized lateral cortical bone width micrometer readings (in mm) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	1.86	0.1654
Treatment	1	27	0.22	0.6416
Treatment*Day	2	54	0.41	0.6642

Table B-83. ANOVA table for lateral cortical bone width micrometer readings (in mm) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	3.30	0.0443
Treatment	1	27	0.09	0.7722
Treatment*Day	2	54	0.20	0.8157

Table B-84. ANOVA table for normalized lateral cortical bone width micrometer readings (in mm) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	3.30	0.0443
Treatment	1	27	0.10	0.7568
Treatment*Day	2	54	0.20	0.8157

Table B-85. ANOVA table for lateral cortical bone width micrometer readings (in mm) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	2.81	0.0690
Treatment	1	27	0.05	0.8278
Treatment*Day	2	54	0.77	0.4666

Table B-86. ANOVA table for normalized lateral cortical bone width micrometer readings (in mm) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	2.81	0.0690
Treatment	1	27	0.28	0.5999
Treatment*Day	2	54	0.77	0.4666

Table B-87. ANOVA table for lateral cortical bone width micrometer readings (in mm) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	0.93	0.4009
Treatment	1	27	1.69	0.2045
Treatment*Day	2	54	0.47	0.6292

Table B-88. ANOVA table for normalized lateral cortical bone width micrometer readings (in mm) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	0.93	0.4009
Treatment	1	27	0.18	0.6750
Treatment*Day	2	54	0.47	0.6292

Table B-89. ANOVA table for medial cortical bone width micrometer readings (in mm) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	4.48	0.0159
Treatment	1	27	0.79	0.3829
Treatment*Day	2	54	1.90	0.1594

Table B-90. ANOVA table for normalized medial cortical bone width micrometer readings (in mm) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	4.48	0.0159
Treatment	1	27	0.33	0.5729
Treatment*Day	2	54	1.90	0.1594

Table B-91. ANOVA table for medial cortical bone width micrometer readings (in mm) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	6.85	0.0022
Treatment	1	27	0.88	0.3574
Treatment*Day	2	54	0.39	0.6823

Table B-92. ANOVA table for normalized medial cortical bone width micrometer readings (in mm) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	6.85	0.0022
Treatment	1	27	0.04	0.8397
Treatment*Day	2	54	0.39	0.6823

Table B-93. ANOVA table for medial cortical bone width micrometer readings (in mm) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	4.26	0.0192
Treatment	1	27	0.92	0.3462
Treatment*Day	2	54	1.28	0.2856

Table B-94. ANOVA table for normalized medial cortical bone width micrometer readings (in mm) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	4.26	0.0192
Treatment	1	27	0.00	0.9974
Treatment*Day	2	54	1.28	0.2856

Table B-95. ANOVA table for medial cortical bone width micrometer readings (in mm) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	1.51	0.2307
Treatment	1	27	0.71	0.4066
Treatment*Day	2	54	1.95	0.1515

Table B-96. ANOVA table for normalized medial cortical bone width micrometer readings (in mm) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	1.51	0.2307
Treatment	1	27	1.81	0.1894
Treatment*Day	2	54	1.95	0.1515

Table B-97. ANOVA table for palmar cortical bone width micrometer readings (in mm) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	0.02	0.9838
Treatment	1	27	0.05	0.8224
Treatment*Day	2	54	0.60	0.5536

Table B-98. ANOVA table for normalized palmar cortical bone width micrometer readings (in mm) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	0.02	0.9838
Treatment	1	27	0.78	0.3853
Treatment*Day	2	54	0.60	0.5536

Table B-99. ANOVA table for palmar cortical bone width micrometer readings (in mm) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	0.05	0.9541
Treatment	1	27	0.35	0.5611
Treatment*Day	2	54	0.51	0.6008

Table B-100. ANOVA table for normalized palmar cortical bone width micrometer readings (in mm) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	0.05	0.9541
Treatment	1	27	0.58	0.4538
Treatment*Day	2	54	0.51	0.6008

Table B-101. ANOVA table for palmar cortical bone width micrometer readings (in mm) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	0.55	0.5814
Treatment	1	27	0.14	0.7124
Treatment*Day	2	54	1.01	0.3715

Table B-102. ANOVA table for normalized palmar cortical bone width micrometer readings (in mm) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	0.55	0.5814
Treatment	1	27	1.25	0.2743
Treatment*Day	2	54	1.01	0.3715

Table B-103. ANOVA table for palmar cortical bone width micrometer readings (in mm) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	10.07	0.0002
Treatment	1	27	0.53	0.4727
Treatment*Day	2	54	0.87	0.4252

Table B-104. ANOVA table for normalized palmar cortical bone width micrometer readings (in mm) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	10.07	0.0002
Treatment	1	27	1.47	0.2356
Treatment*Day	2	54	0.87	0.4252

Table B-105. ANOVA table for dorsal to palmar medullary cavity width micrometer readings (in mm) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	5.74	0.0055
Treatment	1	27	0.29	0.5924
Treatment*Day	2	54	2.05	0.1382

Table B-106. ANOVA table for normalized dorsal to palmar medullary cavity width micrometer readings (in mm) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	5.74	0.0055
Treatment	1	27	2.07	0.1617
Treatment*Day	2	54	2.05	0.1382

Table B-107. ANOVA table for dorsal to palmar medullary cavity width micrometer readings (in mm) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	8.91	0.0005
Treatment	1	27	0.39	0.5387
Treatment*Day	2	54	2.85	0.0667

Table B-108. ANOVA table for normalized dorsal to palmar medullary cavity width micrometer readings (in mm) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	8.91	0.0005
Treatment	1	27	5.42	0.0277
Treatment*Day	2	54	2.85	0.0667

Table B-109. ANOVA table for dorsal to palmar medullary cavity width micrometer readings (in mm) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	1.42	0.2513
Treatment	1	27	0.49	0.4893
Treatment*Day	2	54	1.85	0.1673

Table B-110. ANOVA table for normalized dorsal to palmar medullary cavity width micrometer readings (in mm) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	1.42	0.2513
Treatment	1	27	2.28	0.1428
Treatment*Day	2	54	1.85	0.1673

Table B-111. ANOVA table for dorsal to palmar medullary cavity width micrometer readings (in mm) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	1.41	0.2534
Treatment	1	27	0.40	0.5332
Treatment*Day	2	54	0.55	0.5778

Table B-112. ANOVA table for normalized dorsal to palmar medullary cavity width micrometer readings (in mm) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	1.41	0.2534
Treatment	1	27	0.82	0.3729
Treatment*Day	2	54	0.55	0.5778

Table B-113. ANOVA table for dorsal to palmar bone diameter micrometer readings (in mm) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	7.37	0.0015
Treatment	1	27	0.05	0.8227
Treatment*Day	2	54	1.71	0.1905

Table B-114. ANOVA table for normalized dorsal to palmar bone diameter micrometer readings (in mm) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	7.37	0.0015
Treatment	1	27	1.09	0.3062
Treatment*Day	2	54	1.71	0.1905

Table B-115. ANOVA table for dorsal to palmar bone diameter micrometer readings (in mm) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	10.50	0.0001
Treatment	1	27	0.03	0.8535
Treatment*Day	2	54	1.80	0.1758

Table B-116. ANOVA table for normalized dorsal to palmar bone diameter micrometer readings (in mm) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	10.50	0.0001
Treatment	1	27	1.06	0.3130
Treatment*Day	2	54	1.80	0.1758

Table B-117. ANOVA table for dorsal to palmar bone diameter micrometer readings (in mm) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	17.71	<.0001
Treatment	1	27	0.32	0.5754
Treatment*Day	2	54	1.84	0.1681

Table B-118. ANOVA table for normalized dorsal to palmar bone diameter micrometer readings (in mm) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	17.71	<.0001
Treatment	1	27	1.44	0.2412
Treatment*Day	2	54	1.84	0.1681

Table B-119. ANOVA table for dorsal to palmar bone diameter micrometer readings (in mm) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	31.43	<.0001
Treatment	1	27	0.10	0.7487
Treatment*Day	2	54	0.50	0.6080

Table B-120. ANOVA table for normalized dorsal to palmar bone diameter micrometer readings (in mm) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	31.43	<.0001
Treatment	1	27	0.85	0.3657
Treatment*Day	2	54	0.50	0.6080

Table B-121. ANOVA table for lateral to medial medullary cavity width micrometer readings (in mm) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	0.58	0.5626
Treatment	1	27	0.16	0.6879
Treatment*Day	2	54	1.30	0.2805

Table B-122. ANOVA table for normalized lateral to medial medullary cavity width micrometer readings (in mm) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	0.58	0.5626
Treatment	1	27	0.18	0.6744
Treatment*Day	2	54	1.30	0.2805

Table B-123. ANOVA table for lateral to medial medullary cavity width micrometer readings (in mm) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	0.27	0.7637
Treatment	1	27	0.03	0.8655
Treatment*Day	2	54	0.81	0.4520

Table B-124. ANOVA table for normalized lateral to medial medullary cavity width micrometer readings (in mm) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	0.27	0.7637
Treatment	1	27	0.92	0.3471
Treatment*Day	2	54	0.81	0.4520

Table B-125. ANOVA table for lateral to medial medullary cavity width micrometer readings (in mm) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	0.86	0.4269
Treatment	1	27	0.03	0.8639
Treatment*Day	2	54	1.02	0.3658

Table B-126. ANOVA table for normalized lateral to medial medullary cavity width micrometer readings (in mm) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	0.86	0.4269
Treatment	1	27	0.31	0.5795
Treatment*Day	2	54	1.02	0.3658

Table B-127. ANOVA table for lateral to medial medullary cavity width micrometer readings (in mm) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	1.33	0.2718
Treatment	1	27	0.06	0.8053
Treatment*Day	2	54	1.69	0.1950

Table B-128. ANOVA table for normalized lateral to medial medullary cavity width micrometer readings (in mm) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	1.33	0.2718
Treatment	1	27	1.73	0.1990
Treatment*Day	2	54	1.69	0.1950

Table B-129. ANOVA table for lateral to medial bone diameter micrometer readings (in mm) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	1.53	0.2248
Treatment	1	27	1.58	0.2190
Treatment*Day	2	54	2.52	0.0896

Table B-130. ANOVA table for normalized lateral to medial bone diameter micrometer readings (in mm) at site 1.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	1.53	0.2248
Treatment	1	27	0.02	0.8866
Treatment*Day	2	54	2.52	0.0896

Table B-131. ANOVA table for lateral to medial bone diameter micrometer readings (in mm) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	2.91	0.0632
Treatment	1	27	1.32	0.2606
Treatment*Day	2	54	0.39	0.6777

Table B-132. ANOVA table for normalized lateral to medial bone diameter micrometer readings (in mm) at site 2.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	2.91	0.0632
Treatment	1	27	0.24	0.6276
Treatment*Day	2	54	0.39	0.6777

Table B-133. ANOVA table for lateral to medial bone diameter micrometer readings (in mm) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	1.35	0.3057
Treatment	1	27	1.09	0.2670
Treatment*Day	2	54	0.46	0.6315

Table B-134. ANOVA table for normalized lateral to medial bone diameter micrometer readings (in mm) at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	1.35	0.2670
Treatment	1	27	0.01	0.9190
Treatment*Day	2	54	0.46	0.6315

Table B-135. ANOVA table for lateral to medial bone diameter micrometer readings (in mm) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	1.93	0.1552
Treatment	1	27	0.71	0.4061
Treatment*Day	2	54	1.87	0.1635

Table B-136. ANOVA table for normalized lateral to medial bone diameter micrometer readings (in mm) at site 4.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	1.93	0.1552
Treatment	1	27	0.08	0.7838
Treatment*Day	2	54	1.87	0.1635

Table B-137. ANOVA table for bone index value of micrometer readings at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	15.46	<.0001
Treatment	1	27	0.40	0.5321
Treatment*Day	2	54	2.95	0.0610

Table B-138. ANOVA table for normalized bone index value of micrometer readings at site 3.

Source	numerator df	denominator df	F-Value	Probability > F
Day	2	54	15.46	<.0001
Treatment	1	27	2.03	0.1658
Treatment*Day	2	54	2.95	0.0610

VITA

Katherine Lenore Thomson attended Holy Cross Lutheran School in Dallas, and Pilgrim Lutheran School in Houston. Since starting high school, Albany, Texas has been her home. After graduating from Albany High School, Katherine went to Texas A&M University for 1 ½ years but was unable to make good grades, in part because she was working nights at the Texas A&M Dairy milking cows. Highlighting her brief stay at TAMU was the purchase of her first horse "Red".

After working at a gas station for several months, Katherine was inspired to go back to school. Katherine graduated from Cisco Junior College with an Associate of Science degree, agriculture major in May of 1983. She then attended Tarleton State University, majoring in horse production and management, and graduated with a Bachelor of Science degree in 1985. Highlighting her undergraduate schooling at TSU was serving an internship for Granada Equine Services as a night technician foaling out mares.

Katherine moved back to the farm in Albany and worked for her Grandfather for several years. Red was joined by Rio, Rebel, Misty, Lady Rey, and Chex. Unable to make a living raising horses and cattle, Katherine went back to school.

She earned her Master of Science degree in agriculture from TSU in 1996. After she was turned down by Texas Tech, Katherine accepted the Lord's guidance and went back to TAMU to work on a Ph.D. in equine science under Dr. Potter. While at TAMU she worked as a Teaching Assistant in the equine science department. Inspired by Dr. Hood's physiology class, Katherine took a brief four year break from her Ph.D. program to attend the College of Veterinary Medicine at Texas A&M University where she graduated with her D.V.M. in May 2004. Her fourth year vet school internship was served at the 6666 ranch in Guthrie, Texas. She earned her final degree from TAMU, a Ph.D. in equine science, in August 2005.

Katherine is on faculty at Tarleton State University as an Assistant Professor in the department of animal science teaching equine science and pre-vet classes. She can be reached at T-Box 0070, Stephenville, TX 76402, or at 1751 FM 2482, Albany, TX 76430.