Effect of exercise on thicknesses of mature hyaline cartilage, calcified cartilage, and subchondral bone of equine tarsi

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Objective—To investigate effects of exercise on hyaline cartilage (HC), calcified cartilage (CC), and subchondral bone (SCB) thickness patterns of equine tarsi.

Sample Population—30 tarsi from cadavers of horses with known exercise history.

Procedures—Tarsi were assigned to 3 groups according to known exercise history as follows: pasture exercise only (PE tarsi), low-intensity general-purpose riding exercise (LE tarsi), and high-intensity elite competition riding exercise (EE tarsi). Osteochondral tissue from distal tarsal joints underwent histologic preparation. Hyaline cartilage, CC, and SCB thickness were measured at standard sites at medial, midline, and lateral locations across joints with a histomorphometric technique.

Results—HC, CC, and SCB thickness were significantly greater at all sites in EE tarsi, compared with PE tarsi; this was also true when LE tarsi were compared with PE tarsi. At specific sites, HC, CC, and SCB were significantly thicker in EE tarsi, compared with LE tarsi. Along the articular surface of the proximal aspect of the third metatarsal bone, SCB was thickest in EE tarsi and thinnest in LE tarsi; increases were greatest at sites previously reported to undergo peak strains and osteochondral damage.

Conclusions and Clinical Relevance—Increased exercise was associated with increased HC, CC, and SCB thickness in mature horses. At sites that undergo high compressive strains, with a reported predisposition to osteoarthritic change, there was increased CC and SCB thickness. These results may provide insight into the interaction between adaptive response to exercise and pathological change. (*Am J Vet Res* 2009;70:1477–1483)

Osteoarthritis is a major cause of morbidity, disability, and loss of function in the general human population.[1,2] Overloading of the osteochondral tissues is likely to be a factor in osteoarthritis development, as is exercise-associated osteochondral damage. To improve the understanding of osteoarthritis development, normal osteochondral structure, how it functions, and its response to different types of exercise need to be studied. Horses are susceptible to naturally occurring osteoarthritis and therefore serve as a good model for investigating disease development.[3–5] Osteoarthritis is a major cause of distal tarsal joint pain[6] affecting horses undergoing various exercise regimens.

Abbreviations

AC	Articular cartilage
CC	Calcified cartilage
DistCT	Distal aspect of the central tarsal bone
DistT3	Distal aspect of the third tarsal bone
EE	Elite-exercise group
HC	Hyaline cartilage
LE	Low-exercise group
PE	Pasture-exercise group
ProxMT3	Proximal aspect of the third metatarsal bone
ProxT3	Proximal aspect of the third tarsal bone
SCB	Subchondral bone

Relationships between HC, CC, and SCB are influenced by exercise and age and may be integral to osteoarthritis pathogenesis.[3,7–11,a] An increase in the thickness of either CC or SCB is postulated to lead to potential overloading of the overlying HC.[3,7,8] However, tissue adaptation may also lead to increased thickness of cartilage or SCB.[12,13] An improved understanding of the differences between adaptation and pathological change and what may influence this change is needed.

Thicknesses of HC, CC, and SCB are affected by exercise, age, and location within a joint.[3,4,7–9,14–16,a] Par-

Received October 2, 2008.
Accepted February 2, 2009.
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Supported by The Horse Trust.
The authors thank Ray Wright for performing histologic preparations and Marion Branch for performing magnetic resonance imaging.
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ticular locations are likely to be most susceptible to osteochondral damage. This is related to loading patterns across articular surfaces.[3,4,17–22] In joints of clinically normal individuals, it is anticipated that there are adaptive changes at these sites that could potentially overlap with pathological changes that are subclinical and therefore not associated with pain or lameness. However, sudden overload at these sites may result in the development of pain and lameness. It would therefore be useful to compare osteochondral structure in undamaged joints at these adapted locations with locations less susceptible to injury. Adaptive responses to exercise lead to alterations in thickness of the osteochondral tissues. However, repetitive overloading is associated with osteochondral damage.[4,9,a] Study of osteochondral structure in joints subjected to different exercise types and at locations with and without susceptibility to osteochondral damage could potentially give insight into the distinction between adaptive and pathological change.

Distal tarsal joints in horses are loaded primarily in compression, with peak strains on the dorsal and lateral aspects.[23] On nuclear scintigraphic evaluation, there is also relatively greater radiopharmaceutical uptake on dorsal and lateral aspects of the distal region of the tarsus than elsewhere, supporting increased bone remodeling in these areas.[24] Osteoarthritis of the distal tarsal joints is a major clinical problem in horses.[6] A recent study[25] of relative injury prevalence revealed that osteoarthritis of the tarsus comprised 11.5% of lameness in general-purpose riding horses, 16.8% in elite show jumpers, and 15% in endurance horses. The dorsomedial aspect of the centrodistal (distal intertarsal) joint and the dorsolateral aspect of the tarsometatarsal joint are frequent sites of osteochondral damage in horses.[26,27] Equine centrodistal and tarsometatarsal joints provide useful osteochondral tissues for study because the exercise history of horses is often known and the size of the bones enables study of multiple locations within a joint.

The purposes of the study reported here were to measure AC, HC, CC, and SCB thickness on DistCT, ProxT3, DistT3, and ProxMT3 at medial, midline, and lateral sites in tarsi from horses without hind limb lameness and with a known exercise history and to compare patterns of cartilage and SCB thickness among exercise groups. It was hypothesized that high-level competition exercise is associated with thicker AC, HC, CC, and SCB thickness at dorsal sites of the tarsus than low-intensity exercise and pasture exercise only.

Materials and Methods

Sample collection—Thirty tarsi were collected from 17 horses euthanatized for reasons unrelated to this study. Horses were euthanatized by IV injection of an overdose of secobarbital sodium and cinchocaine hydrochloride solution.[b] Tarsi were included in the study if horses had no history or clinical evidence of hind limb lameness and had no radiographic evidence of an abnormality (alteration of opacity of SCB or cancellous bone, endosteal irregularity, reduction in joint space width, osteophyte formation, periarticular periosteal new bone formation, or cystic lesion). Tarsi were excluded if they were collected from horses with a previous history of hind limb lameness. No horses had signs of lameness at the time of euthanasia. Tarsi were collected within 6 hours of death and stored frozen at –20°C. The study was approved by the Ethical Review Committee of the Animal Health Trust.

Exercise groups—Thirty cadaver tarsal joints from 17 horses were assigned to 1 of 3 groups on the basis of the known exercise history of the horses. Pasture-exercise group tarsi (n = 10) were collected from 5 mature female Thoroughbreds (mean, 14 years old; range, 10 to 19 years old), which had been restricted to pasture exercise only for a mean period of 13 years (range, 9 to 16 years) after having raced at 2 years of age. Low-exercise group tarsi (n = 10) were collected from 6 mature horses (mean, 11.3 years old; range, 7 to 15 years old) that had a history of low-intensity general-purpose riding exercise, defined as hacking and unaffiliated competition. There were 6 left tarsi and 4 right tarsi from 4 geldings and 2 mares that were of the following breeds: 2 Thoroughbred crossbred horses and 4 warmblood horses. Elite-exercise group tarsi (n = 10) were collected from 6 mature horses (mean, 12.9 years old; range, 10 to 17 years old) that had a history of high-intensity elite competition riding exercise. Elite competition was defined as high-level national or international show jumping, dressage, or 3-day eventing competition, which includes jumping, galloping, tight turns, and gymnastic-type exercises. There were 5 left tarsi and 5 right tarsi from 6 geldings that were of the following breeds: 2 Thoroughbred crossbred horses and 4 warmblood horses.

Histologic preparation—Frozen tarsi were clamped with a heavy-duty vice to grip the distal portion of the tibia and the calcaneus. By use of a large band saw,[c] the distal tarsal joints were cut into sagittal slices that were 4 to 5 mm thick and detached by cutting in a transverse plane above the talocalcaneal-centroquartal (proximal intertarsal) joints. To define medial (30%), midline (50%), and lateral (70%) sites on each tarsus for sectioning, sites previously[28] defined on sagittal 3-D T1-weighted spoiled gradient echo magnetic resonance images were translated onto the frozen tarsus by means of image cross-referencing. By use of a small band saw,[d] the chosen medial, midline, and lateral slices were cut in a transverse and then a dorsal plane to produce dorsal sections through the tarsometatarsal joint that were half the dorsal to plantar depth of the third tarsal bone.

Sections were fixed in individually labeled containers containing neutral-buffered 10% formalin for 10 days. Sections were removed from the formalin, washed to remove bone dust, and placed in a rapid decalcifier[e] for 2 days. When removed from the rapid decalcifier, sections were placed under running tap water for 1 day. They were then dehydrated in 70%, 90%, and 3 changes of 100% ethyl alcohol and then 3 changes of xylene and 3 changes of molten histologic wax[f] on an automatic tissue processor[g] and embedded in blocks of paraffin wax.

By use of a heavy-duty rotary microtome,[h] sections were cut at a thickness of 6 μm and then mounted on

gelatin-coated slides produced by dipping cleaned slides in hot gelatin[i] and drying them in an oven at 37°C. One slide was stained with Harris' H&E stain and the other with 1% toluidine blue and then mounted in a mixture of distyrene, a plasticizer, and xylene mountant.[j]

Histomorphometry—Measurements were performed on digital images that were obtained with a microscope[k] linked to a digital camera.[l] Thickness measurements of AC, HC, CC, and SCB were obtained by use of a histomorphometric technique as previously described.[3,29] To briefly summarize, measurements were obtained at standard sites at dorsal locations that were 15% of the dorsal to plantar depth of the third tarsal bone on DistCT, ProxT3, DistT3, and ProxMT3. Dorsal measurements were made at medial, midline, and lateral sites, which were 30%, 50%, and 70% of the medial to lateral width of the third tarsal bone, respectively. All measurements were obtained perpendicular to the articular surface at standard sites by use of image analysis software.[m] Repeatability at each site was assessed by use of 10 repeated measurements/site for 10 limbs. Final measurements were obtained when the coefficient of variance was < 1% to ensure a high level of accuracy.

Statistical analysis—Descriptive statistics were used to obtain age, height, and weight distributions of horses of the exercise groups. A Shapiro-Wilk test was used to assess whether data were parametric or nonparametric. A 1-way ANOVA (for parametric data) or a Mann-Whitney *U* test (for nonparametric data) was used to compare AC, HC, CC, and SCB thicknesses and proportions of HC and CC among exercise groups, taking account of repeated measures. All analyses were performed by use of statistical analysis software[n] with the significance level set at $P < 0.05$.

Results

Comparison of full AC thickness among exercise groups—Mean ± SD full AC thickness in EE tarsi was significantly greater than that in PE tarsi at all sites (**Table 1**). Full AC thickness was consistently greater in EE tarsi, compared with LE tarsi, but this difference was only significant ($P = 0.003$) at the lateral site of ProxMT3. Full AC thickness was significantly greater in LE tarsi, compared with PE tarsi, at all sites except at medial sites of DistCT and DistT3.

Comparison of HC thickness among exercise groups—Mean ± SD thickness of the HC in EE tarsi was significantly greater than that in PE tarsi at all sites (**Table 2**). Thickness of the HC was greater at the lateral site of DistT3 in EE tarsi, compared with LE tarsi, but this difference was not significant ($P = 0.066$). Thickness of the HC was significantly greater in LE tarsi, compared with PE tarsi, at all sites except at midline and lateral sites of DistT3.

Comparison of CC thickness among exercise groups—Mean ± SD thickness of the CC in EE tarsi was significantly thicker than that in PE tarsi at all sites, except at medial sites of DistCT and ProxT3 and the midline site of DistCT (**Table 3**). Thickness of the CC was significantly greater at the medial site of DistT3 ($P = 0.028$) and lateral site of ProxMT3 ($P = 0.003$) in EE tarsi, compared with LE tarsi. Thickness of the CC was significantly greater in LE tarsi, compared with PE tarsi, at all sites except at medial sites of DistCT, ProxT3, and DistT3 and the midline site of DistCT.

Comparison of SBC thickness among exercise groups—Mean ± SD thickness of SCB in EE tarsi was significantly thicker than that in PE tarsi at all sites (**Table 4**). Thickness of the SCB was significantly greater at medial sites of DistCT ($P = 0.002$), ProxT3 ($P = 0.005$), DistT3 ($P = 0.025$), and ProxMT3 ($P < 0.001$); midline site of ProxMT3 ($P = 0.010$); and lateral sites of DistCT ($P = 0.046$), ProxT3 ($P = 0.010$), and ProxMT3 ($P = 0.040$) in EE tarsi, compared with LE tarsi. Thickness of the SCB was significantly greater in PE tarsi, compared with LE tarsi, at medial sites of DistCT ($P = 0.037$) and ProxMT3 ($P = 0.027$). Along the articular surface of ProxMT3, SCB was thickest laterally and thinnest at the midline site for all 3 exercise groups. Overall, SCB was thickest in EE tarsi at all 3 sites; SCB was thinnest in LE tarsi.

Comparisons of cartilage and SCB thicknesses among exercise groups at sites predisposed to osteoarthritis—At the medial aspect of the centrodistal joint (ie, medial sites of DistCT and ProxT3), HC and CC were thicker in EE and LE tarsi, compared with PE tarsi (Tables 2 and 3). However, SCB was thickest in EE tarsi and thinnest in LE tarsi (Table 4). The overall thickness of the osteochondral unit was greatest in EE tarsi and smallest in LE tarsi (**Table 5**).

Table 1—Mean ± SD full thickness (mm) of AC of the distal tarsal joints from horses with a known exercise history (n =10 tarsi/group).

Site	Location	PE tarsi	LE tarsi	EE tarsi
Medial	DistCT	0.697 ± 0.257*	0.877 ± 0.143	0.928 ± 0.128*
	ProxT3	0.675 ± 0.252*†	0.892 ± 0.162†	0.946 ± 0.250*
	DistT3	0.657 ± 0.247*	0.819 ± 0.138	0.936 ± 0.146*
	ProxMT3	0.562 ± 0.189*†	0.890 ± 0.118†	0.930 ± 0.101*
Midline	DistCT	0.641 ± 0.243*†	0.832 ± 0.010†	0.851 ± 0.151*
	ProxT3	0.582 ± 0.232*†	0.880 ± 0.180†	0.918 ± 0.215*
	DistT3	0.628 ± 0.256*†	0.854 ± 0.144†	1.004 ± 0.207*
	ProxMT3	0.639 ± 0.240*†	0.902 ± 0.209†	0.984 ± 0.234*
Lateral	DistCT	0.489 ± 0.191*†	0.798 ± 0.144†	0.807 ± 0.105*
	ProxT3	0.571 ± 0.236*†	0.801 ± 0.149†	0.818 ± 0.128*
	DistT3	0.547 ± 0.195*†	0.880 ± 0.118†	0.886 ± 0.114*
	ProxMT3	0.536 ± 0.207*†	0.779 ± 0.117†‡	0.984 ± 0.151*‡

*Significant difference ($P < 0.05$) between PE and EE tarsi. †Significant difference ($P < 0.05$) between PE and LE tarsi. ‡Significant difference ($P < 0.05$) between LE and EE tarsi.

At the lateral aspect of the tarsometatarsal joint (ie, lateral sites of DistT3 and Prox MT3), HC and CC were thicker in EE and LE tarsi, compared with PE tarsi (Tables 2 and 3). The SCB was thickest in PE and EE tarsi at ProxMT3 (Table 4). The overall thickness of the osteochondral unit was greatest in LE and EE tarsi at DistT3. At ProxMT3, the osteochondral unit was greatest in EE tarsi and smallest in LE tarsi (Table 5).

Table 2—Mean ± SD thickness (mm) of HC of the distal tarsal joints from horses with a known exercise history.

Site	Location	PE tarsi	LE tarsi	EE tarsi
Medial	DistCT	0.385 ± 0.139*†	0.505 ± 0.144†	0.539 ± 0.130*
	ProxT3	0.364 ± 0.168*†	0.567 ± 0.143†	0.594 ± 0.208*
	DistT3	0.371 ± 0.148*†	0.486 ± 0.048†	0.506 ± 0.142*
	ProxMT3	0.349 ± 0.148*†	0.512 ± 0.075†	0.494 ± 0.084*
Midline	DistCT	0.330 ± 0.111*†	0.485 ± 0.105†	0.465 ± 0.121*
	ProxT3	0.335 ± 0.132*†	0.519 ± 0.172†	0.509 ± 0.119*
	DistT3	0.360 ± 0.160*	0.453 ± 0.067	0.522 ± 0.129*
	ProxMT3	0.363 ± 0.147*†	0.475 ± 0.010†	0.526 ± 0.130*
Lateral	DistCT	0.348 ± 0.143*†	0.475 ± 0.083†	0.504 ± 0.099*
	ProxT3	0.382 ± 0.145*†	0.484 ± 0.092†	0.479 ± 0.083*
	DistT3	0.349 ± 0.170*	0.482 ± 0.059	0.544 ± 0.076*
	ProxMT3	0.334 ± 0.156*†	0.505 ± 0.107†	0.494 ± 0.059*

See Table 1 for key.

Table 3—Mean ± SD thickness (mm) of CC of the distal tarsal joints from horses with a known exercise history.

Site	Location	PE tarsi	LE tarsi	EE tarsi
Medial	DistCT	0.312 ± 0.130	0.372 ± 0.134	0.389 ± 0.062
	ProxT3	0.312 ± 0.112	0.325 ± 0.157	0.352 ± 0.091
	DistT3	0.304 ± 0.146*	0.330 ± 0.160‡	0.430 ± 0.057*‡
	ProxMT3	0.213 ± 0.099*†	0.378 ± 0.148†	0.436 ± 0.067*
Midline	DistCT	0.311 ± 0.178	0.347 ± 0.088	0.386 ± 0.089
	ProxT3	0.247 ± 0.123*†	0.361 ± 0.104†	0.409 ± 0.120*
	DistT3	0.268 ± 0.132*†	0.401 ± 0.136†	0.482 ± 0.105*
	ProxMT3	0.276 ± 0.116*†	0.427 ± 0.155†	0.458 ± 0.147*
Lateral	DistCT	0.141 ± 0.078*†	0.323 ± 0.087†	0.303 ± 0.085*
	ProxT3	0.189 ± 0.113*†	0.317 ± 0.078†	0.339 ± 0.086*
	DistT3	0.193 ± 0.081*†	0.398 ± 0.094†	0.342 ± 0.078*
	ProxMT3	0.202 ± 0.092*†	0.274 ± 0.117†‡	0.455 ± 0.121*‡

See Table 1 for key.

Table 4—Mean ± SD thickness (mm) of SCB of the distal tarsal joints from horses with a known exercise history.

Site	Location	PE tarsi	LE tarsi	EE tarsi
Medial	DistCT	4.73 ± 1.63*†	3.25 ± 1.36†‡	7.07 ± 2.78*‡
	ProxT3	5.07 ± 2.62*	4.16 ± 1.80‡	8.02 ± 3.36*‡
	DistT3	3.85 ± 1.38*	3.82 ± 1.30‡	6.08 ± 2.53*‡
	ProxMT3	10.12 ± 3.26*†	6.52 ± 3.43†‡	12.58 ± 3.13*‡
Midline	DistCT	3.36 ± 2.32*	3.93 ± 2.81	6.22 ± 4.01*
	ProxT3	4.62 ± 2.25*	5.36 ± 1.33	7.81 ± 5.14*
	DistT3	3.93 ± 2.76*	5.09 ± 2.68	6.82 ± 3.41*
	ProxMT3	6.87 ± 3.83*	5.14 ± 2.01‡	10.62 ± 5.24*‡
Lateral	DistCT	6.10 ± 1.81*	6.40 ± 1.95‡	8.39 ± 2.20*‡
	ProxT3	6.26 ± 2.19*	5.21 ± 2.31‡	9.23 ± 3.79*‡
	DistT3	7.46 ± 3.24*	9.30 ± 3.02	9.99 ± 3.46*
	ProxMT3	10.82 ± 5.36*	9.71 ± 4.66‡	14.62 ± 4.94*‡

See Table 1 for key.

Table 5—Mean ± SD overall thickness (mm) of the osteochondral unit at sites predisposed to osteoarthritis in the distal tarsal joints from horses with a known exercise history.

Site	Location	PE tarsi	LE tarsi	EE tarsi
Medial	DistCT	5.45 ± 1.50*	4.13 ± 1.41‡	8.00 ± 2.74*‡
	ProxT3	5.77 ± 2.59*	5.05 ± 1.78	8.97 ± 3.33*
Lateral	DistT3	8.02 ± 3.29*	10.18 ± 3.00	10.93 ± 3.70*
	ProxMT3	11.36 ± 5.42*†	10.51 ± 4.73†‡	16.29 ± 4.75*‡

See Table 1 for key.

Discussion

Results from this study supported the hypothesis that a history of high-intensity exercise is associated with greater HC, CC, and SCB thickness than either low-intensity exercise or pasture exercise only. In general, the overall thickness of the cartilage and SCB was lowest in pasture-exercised horses, slightly greater in low-intensity exercised horses, and maximal in horses with a history of elite competition exercise.

Findings of our study are likely to reflect a chondrocyte anabolic response to exercises[30–32] at the level both of HC and CC and suggested that this response can occur in mature joints. Increased HC thickness with exercise is reported to occur in immature joints of horses,[3,15,16] dogs,[9] and humans,[14] but it has been suggested that an increase in thickness is not observed with increased loading in mature cartilage of elite human athletes.[33–35] This species difference between horses and humans may reflect the greater loads placed on equine limb joints, the relatively longer athletic career of horses, and the fact that horses spend a much greater proportion of their life standing and ambulating, compared with human athletes. Greater CC thickness with exercise than rest has previously been reported in immature joints of horses[3] and immature dogs[9] undergoing treadmill exercise. It has been suggested that variations in loading across the articular surface are reflected by local variations in CC thickness.[3,8,15] Findings in our study revealed corresponding increases in all the tissues of the mature osteochondral unit in a noninterventional, overground (and not treadmill) situation. General-purpose and competition riding horses are not ridden or trained until skeletal maturity, so it is likely that increase cartilage thickness was induced by functional adaptation after maturity.

In the present study, SCB thickness increased with increased exercise intensity, which supports findings in other equine studies[4,16,28,36,37] and those in others species.[9,12] Small numbers of high strains stimulate bone remodelling,[38,39] so it is not surprising that elite competition exercise would be associated with greater SCB thickness than either low-intensity exercise or pasture exercise only. However, there were some sites where tarsi from horses with a history of pasture exercise only had thicker SCB than tarsi from horses with a history of low-intensity general-purpose riding exercise. It is possible that there was residual SCB thickness from a previous period in which only pasture-exercise horses had raced at 2 years of age. This would suggest that SCB thickness is retained without changing over a period of at least 9 years. In female human athletes commencing squash training in childhood or after puberty,[40] it was found that players that had started squash training before puberty had greater bone mineral density and content, compared with players that had started training after puberty. Also in that study,[40] it was noted that the arm that held the racquet (playing extremity) had higher bone mineral density and bone mineral content, compared with that of the nonplaying extremity.

Alternatively, the thicker SCB in some sites of tarsi from horses with a history of pasture exercise only, compared with tarsi from horses with a history of low-intensity general-purpose riding exercise, could be related to different types of activities undertaken by the 2 groups. At pasture, exercise is self-controlled and generally consists of continual low strains interspersed with short periods of high strains resulting from variable terrain or as horses run and twist, which could stimulate bone remodelling.[38] In contrast, low-intensity general-purpose riding exercise, particularly on soft, consistent training surfaces, is more likely to induce consistent low to moderate strains in the distal tarsal joints. We have previously shown that different types of exercise are associated with different patterns of SCB thickness across the distal tarsal articular surfaces, likely reflecting different patterns of mechanical loading exerted by different types of exercise.[41] In low-intensity general-purpose riding exercise, horses are often restricted to stables for a proportion of each day, in contrast to the continual movement at pasture, which could also have effects on bone remodelling. Continuous pasture exercise has been shown to increase bone mineral density, compared with box stall confinement and regulated exercise, in young horses.[42]

Peak strains have been reported to occur on the dorsolateral aspect of the distal tarsal joints and ProxMT3.[24] At the level of the tibia, there are primarily medial strains,[43] which are transferred from a medial to lateral direction through the tarsus. Findings in our study revealed greater SCB thickness on the medial aspect of the centrodistal joint and the lateral aspect of the tarsometatarsal joint, which is probably an adaptation to these loading patterns. Focal loading is shown to cause focal increases in AC, CC, and SCB thickness in stifle joints of immature dogs,[9] and patterns of CC thickness across human joints have been suggested to reflect local increases in loading.[8] The lateral site of ProxMT3 is an area that is exposed to high strains and is predisposed to osteochondral damage. At this site, EE tarsi in the present study had thinner HC, thicker CC, and thicker SCB than did LE tarsi. The HC, CC, and SCB of EE tarsi were thicker than those of PE tarsi. It has been suggested that in osteoarthritic joints, there is advancement of the tidemark with a relative increase in CC and decrease in HC thickness.[3,9] Although physiologic loads can lead to anabolic chondrocyte responses, mechanical overloading may result in catabolism or chondrocyte death.[15,44–48] It is therefore possible that adaptive increases in CC thickness, and the associated increase in structural stiffness, could ultimately become a contributor to HC degeneration when a threshold is exceeded.[3] It has been suggested that AC is part of the secondary center of ossification that is reactivated under certain types of repetitive loading in adults. New cartilage is formed and is calcified, causing an increase in CC and SCB thickness but not HC thickness.[49]

This study was limited with respect to sample size. However, as a study in which limbs from horses with a known exercise history were used, it has benefits of examining true in vivo adaptation. Moreover, it included horses euthanatized for clinical reasons as opposed to elective euthanasia as part of an experimental study. The exercise history was defined by the owner or rider and not as part of a controlled exercise program; therefore, there are likely to be some variations in exact exercise program and duration among horses. However, the

sample selection criteria were strictly defined to ensure clear differences in amounts of exercise among the 3 groups. There were differences in distribution of breed and sex in the 3 groups, which may have confounded our results.

Our findings suggested that thickness of both cartilage and SCB was affected by exercise intensity in mature horses and that increased exercise intensity was associated with increased thickness. They also supported differences in stimuli for adaptation in cartilage and SCB. From our results, we suggest that a site that undergoes high compressive strains, with predisposition to osteoarthritis, may have increased CC and SCB thickness relative to HC thickness. These findings support further investigation into the threshold between adaptive response to exercise and pathological change.

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