**EQUINE CARTILAGE AND SUBCHONDRAL BONE CHANGE TOGETHER IN RESPONSE TO HIGH INTENSITY EXERCISE**

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**Introduction:** Dorsal carpal osteochondral injury is a major cause of reduced performance in Thoroughbred horses in training. In both humans and horses it is clear that the integrity of articular cartilage and subchondral bone are intimately related, although there is controversy about their relative importance in development of osteoarthritis. Calcified cartilage provides the mechanical link between soft hyaline cartilage and stiff subchondral bone. However, few investigations have studied the response of calcified cartilage and subchondral bone together. In the equine middle carpal joint, increased third carpal subchondral bone stiffness and a loss of hyaline cartilage stiffness at common sites of clinical lesions has been demonstrated in response to training. We hypothesised that there would be increased thickness and modelling of the calcified tissues of the joint surface associated with training and at sites of high, intermittent weight bearing (dorsal locations).

**Methods:** Twelve untrained Thoroughbred horses (18-21 months old) were paired according to size. One of each pair was randomly assigned to group 1 (n=6) or group 2 (n=6). Institutional review board approval was obtained for this study. Group 1 (high intensity) horses underwent a 19 week progressive high intensity training regimen on a high speed treadmill. Training consisted of 3 sessions per week. Sessions were 4,800 m at 10 m/s; 3 periods of 800 m at 13 m/s or 2 periods of 1300 m at 11 m/s. Each horse also underwent 40 minutes daily walking, and 20 minutes trotting 3 days per week. Group 2 (low intensity) underwent daily walking only. Left carpi were collected immediately following euthanasia at the end of the program. Eight sites were identified per joint: radial dorsal, radial palmar, intermediate dorsal, intermediate palmar, third carpal medial facet dorsal, medial facet palmar, lateral facet dorsal, lateral facet palmar. Osteochondral specimens were obtained from each test site. Samples were decalcified and embedded in paraffin for assessment of cartilage thickness. For assessment of calcified cartilage and subchondral bone, undecalcified samples were embedded in LR White resin. Sections underwent toluidine blue and von Kossa staining. Histomorphometric measurements of thickness of total cartilage, hyaline layer, cartilage calcified zone and subchondral bone were performed at each site (2). Relative osteoid surface was calculated as a proportion of the total bone surface. Analysis of variance procedures were used to compare results between exercise groups, and between dorsal (high, intermittent load) and palmar (lower, more continual load) locations (p<0.05).

**Results:**

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Topographical and exercise related differences were found (Table 1). The most marked changes were at the radial dorsal site in group 1. **Cartilage - Group 1 horses had significantly thicker total cartilage (p<0.0001) and calcified zone (p<0.0001) than group 2. There was no difference in hyaline layer thickness between exercise groups. Within each exercise group all dorsal sites had thicker calcified cartilage (p<0.0003) than palmar sites. There was no difference in hyaline cartilage or total cartilage thickness between dorsal and palmar sites.**

**Subchondral Bone - Group 1 horses had significantly thicker subchondral bone (p<0.0001) than group 2, and this difference was most marked at dorsal sites. Within each exercise group all dorsal sites had thicker subchondral bone than palmar sites (p<0.001). Microcracks were not observed in either exercise group. Relative osteoid surface was significantly greater in group 1 than group 2 at dorsal sites (p<0.001). At palmar sites differences between exercise groups were not observed. The greatest relative osteoid surface was found at dorsal sites in group 1.**

**Discussion:**

Marked topographical and exercise related differences were found within the cartilage and subchondral bone, showing a combined response of the osteochondral unit to loading. The study is however, limited by being confined solely to the static bone indices. Parallel topographical responses in the cartilage and subchondral bone were observed at sites of high load bearing (dorsal sites). Strenuous exercise led to increased subchondral bone modeling and calcified cartilage thickness, particularly at dorsal sites, demonstrating a combined response of both tissues to training. It has been suggested that a gradual stiffness transition between cartilage and bone is provided by the calcified layer. We have previously demonstrated loss of cartilage stiffness in radial and third carpal articular surfaces from horses in group 1 (1). From our findings in this study, it is evident that there is increased calcified cartilage thickness at sites where there was reduced cartilage stiffness and significant subchondral bone modeling. It is feasible therefore, that by increasing calcified cartilage thickness with exercise, the stiffness gradient of the articular surface may be maintained in the face of reduced hyaline cartilage stiffness and increased subchondral bone stiffness.

Under pathological conditions, mineralisation of articular cartilage progresses towards the surface. If the tidemark advances significantly into the hyaline cartilage in the presence of subchondral bone sclerosis, the biomechanical shock absorbing capacity of the tissue may be impaired. Although increased calcified cartilage thickness may initially reduce cartilage strains, excessive tidemark advancement may eventually contribute to cartilage destruction. At the dorsal radial carpal location in the high intensity exercise group, there was greatest proportion of calcified cartilage, and least hyaline cartilage compared to all other sites. This location is the most common site of osteochondral pathology in the equine middle carpal joint, and was a site where noted cartilage fibrillation in the horses used in this study (1). At this site there was no evidence of subchondral bone pathology, but a marked modeling response was observed. From our findings we would suggest that tidemark advancement associated with high intensity training represents a physiologic response, but may also be associated with early pathology at intensely loaded sites. Further work is required to determine the demarcation between a physiologic and pathologic response.

These findings indicate that high intensity exercise leads to greater calcified zone depth and increased bone modeling, and that these responses are maximal at sites subjected high loads. By increasing calcified cartilage thickness with exercise, the stiffness gradient of the articular surface may be maintained in the face of alterations in hyaline cartilage or subchondral bone stiffness. However, excessive tidemark advancement may eventually contribute to the degenerative process.

**Table 1. Data in mean±S.D.**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Dorsal sites</th>
<th>Palmar sites</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cartilage thickness (μm)</td>
<td>766±158</td>
<td>673±148</td>
</tr>
<tr>
<td>Hyaline cartilage thickness (μm)</td>
<td>495±122</td>
<td>510±135</td>
</tr>
<tr>
<td>Calcified cartilage thickness</td>
<td>271±73</td>
<td>163±49</td>
</tr>
<tr>
<td>Subchondral bone thickness (μm)</td>
<td>1057±362</td>
<td>670±305</td>
</tr>
<tr>
<td>Relative osteoid surface (%)</td>
<td>2.05±1.04</td>
<td>1.20±0.86</td>
</tr>
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</table>

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**References:**


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