Original Research

# Enthesophytosis and Impingement of the Dorsal Spinous Processes in the Equine Thoracolumbar Spine 

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#### Abstract

Impingement of the dorsal spinous processes (DSPs) is a common cause of pain and poor performance in sport horses, but there is limited information regarding regional differences in the prevalence and severity of DSP osseous lesions in the equine thoracolumbar spine. It was hypothesized that lesion severity would increase with horse age and height, and that severe lesions would be more prevalent in the mid-caudal thoracic region. The thoracolumbar spines of 33 horses were removed postmortem, disarticulated, and boiled out. The thoracic and lumbar DSPs were examined for the presence of proliferative or lytic osseous lesions of the DSPs. Age and height of the horses were recorded, and severity of pathologic changes at each vertebral level was scored using an ordinal grading system (grades 0-3) and a continuous visual analog scale (VAS). Osseous lesions of the DSPs were present at every vertebral level from C7T1 to L6-S1, and 70\% of horses had at least one lesion of severity grade 2 or higher. Grade 3 lesions were found in the cranial thoracic (T2-T4), mid-thoracic to cranial lumbar (T11-L1) and mid-lumbar (L4-L5) segments. Analysis of VAS data using analysis of variance indicated that increasing age and height were associated with more severe osseous lesions ( $P<.001$ ). DSP osseous lesions occur frequently in horses with more severe lesions in the cranial thoracic, mid to caudal thoracic, and mid-lumbar regions. Lesions in the cranial thoracic and lumbar regions present a challenge for diagnostic imaging and may be underdiagnosed clinically.


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## 1. Introduction

Pathologic changes in the vertebrae and associated soft tissues are recognized as causes of back pain and poor performance in athletic horses [1-3]. Osseous lesions that have been associated with equine back pain include vertebral body spondylosis, impingement or overriding of the dorsal spinous processes (DSPs), and osteoarthritis of the synovial intervertebral articulations (SIAs) [1-10].

[^0]Impingement of the DSPs has been reported to be the most common osseous pathology of the equine thoracolumbar vertebrae $[1,5,11,12]$. However, osseous pathology of the DSPs may be present without signs of back pain [11,13,14].

The size, shape, and orientation of the DSPs show regional differences along the length of the thoracolumbar spine [15]. A rudimentary DSP appears on the last cervical (C7) vertebra with the succeeding thoracic vertebrae having progressively longer DSPs to the level of T4 or T5 beyond which they become shorter to the level of T12 [15]. The T12 DSP marks the transition between the narrower processes that form the withers and the wider processes of the more caudal vertebrae [16]. The DSP on T16 is vertically oriented and indicates the juncture between the caudally
sloping DSPs of the more cranial vertebrae and the cranially sloping DSPs of the more caudal vertebrae; for this reason, T16 is called the anticlinal vertebra. Attachment sites of the supraspinous and interspinous ligaments, which limit distraction of the DSPs, m. multifidi, which move and stabilize the intervertebral joints, and the thoracolumbar fascia are potential sites for the development of enthesophytes. It has been suggested that adjacent DSPs should be separated by a distance of at least $4 \mathrm{~mm}[6,17]$ and, if there is insufficient separation of adjacent spines, impingement or overriding of the DSPs with bone-on-bone contact may occur, leading to proliferative or lytic osseous lesions.

Mechanical loading of the spine due to ground reaction forces transmitted through the limb girdles, gravitational and inertial effects of the horse's visceral mass, and the rider's weight are likely to affect the regional susceptibility to impingement of the DSPs. The thoracolumbar spine as a whole is supported peripherally by the limbs, but the midthoracic region has relatively poor mechanical support and becomes lordotic during locomotion [18], which approximates the DSPs in this area. Therefore, it is not surprising that most clinically significant lesions of the DSPs have been reported to involve the region from T13 to T18 [3,9,13].

Necropsy studies are useful to characterize details of the type and severity of DSP osseous lesions that may not be apparent radiographically or ultrasonographically. Previous necropsy studies of the equine DSPs have focused on the caudal thoracic and lumbar regions of Thoroughbred racehorses $[2,5,19,20]$. Prevalence of osseous lesions at different spinal levels has been correlated with thoracolumbar biomechanics to better understand the regional distribution of lesions [8]. The present study expands knowledge by evaluating osseous lesions of the DSPs along the entire length of the thoracolumbar spine in horses of various ages and sizes. It was hypothesized that lesion severity would increase with age and height of the horse and that severe lesions would be more prevalent in the mid to caudal thoracic region, which undergoes a relatively large range of motion in extension as a consequence of having poor mechanical support.

## 2. Materials and Methods

### 2.1. Specimens

The spines were from 33 horses euthanized for reasons other than primary back pain. Twelve breeds were represented: Thoroughbred (9), Quarter Horse (6), Pony (5), Warmblood (3), Arabian (2), Appaloosa (2), Missouri Fox Trotter (1), Percheron (1), Belgian (1), Morgan (1), Morgan cross (1), and Palomino (1). Ages ranged from 2 to 29 years, and individuals were categorized as young ( 17 horses aged $<15$ years) or old ( 16 horses $\geq 15$ years). Horses were also classified by height at the withers as small ( 13 horses $<152 \mathrm{~cm}$ ) or large ( 20 horses $\geq 152 \mathrm{~cm}$ ).

### 2.2. Preparation of Specimens

The entire thoracolumbar spine from C 7 to the sacrum was removed intact. After dissecting away the soft tissues, the vertebrae were disarticulated, boiled in water, soap, and
bleach for 15 hours to remove any remaining soft tissue, and then soaked in a solution of $34.5 \%$ isopropyl alcohol (99\%), $1.7 \%$ hydrogen peroxide ( $30 \%$ ), $0.6 \%$ ammonium hydroxide, and $63.2 \%$ water for at least 2 days to remove residual soft tissues from the bone. The vertebrae were air-dried before being evaluated.

The location, size, and shape of DSP lytic and proliferative osseous lesions were recorded. Because impingement involves contact between the articular processes of adjacent vertebrae, a vertebral level was defined as the caudal surface of one DSP and the cranial surface of the next, more caudal, DSP. Twenty-four vertebral levels were examined from C7-T1 to L6-S1 for osseous lesions indicative of impingement or the presence of enthesophytosis. The lesions were graded both on an ordinal scale and on a continuous visual analog scale (VAS).

### 2.3. Grade Assignment

A noncumulative grading system as described by Stubbs et al [20] and based on methods used previously $[3,5,8]$ was used. For each spinal level, the caudal surface of one DSP and the cranial surface of the adjacent, more caudal DSP were graded separately for severity of osseous lesions on a scale from 0 to 3 according to the following criteria: Grade 0: no lesions, smooth periosteum with no evidence of direct contact between successive DSPs; Grade 1: mild osseous lesion $<2 \mathrm{~mm}$ in length, which might include some impingement of DSPs, mild periosteal proliferation, and mild eburnation but no lysis; Grade 2: moderate osseous degenerative changes 2 to 4 mm in length that might include moderate impingement of, or interference with, the adjacent DSP, moderate to large active periosteal proliferation and/or lysis, moderate eburnation and moderate active remodeling; and Grade 3: severe osseous degenerative changes with bony proliferation $>4 \mathrm{~mm}$ long and that might show evidence of severe impingement of adjacent DSPs, extensive periosteal proliferation and/or lysis, marked eburnation, marked active bone remodeling, and ankylosis. Examples of lesions within each grade are shown in Fig. 1. After grading the lesions on the adjacent caudal and cranial aspects of adjacent DSPs, the higher of the assigned grades was used to represent that level in the statistical analysis.

### 2.4. Visual Analog Scale

The lesions between each pair of adjacent DSPs were also scored using a VAS. The VAS scores were assigned by the same evaluator as the ordinal grades but on a different occasion and with the evaluator blinded to the previously assigned grades. Continuous numerical data were obtained using a $20-\mathrm{cm}$ line, the left edge of which represented no lesion, and the right edge represented an extremely severe lesion. A mark was made along the line to represent the overall severity of the DSP osseous lesions at that spinal level. The distance from the left side of the line to the mark was measured in centimeters to obtain the VAS score for DSP osseous pathology at each spinal level. The VAS scores have the advantage of allowing a larger range of pathologic changes to be incorporated into the score.


Fig. 1. Examples of grade assignment for osseous lesions of the dorsal spinous processes (DSPs) in the thoracic region. All photographs show the dorsocaudal aspect of the DSP. Left to right: grade 0: no lesions with smooth periosteum, no proliferation or lysis; grade 1: mild periosteal proliferation (arrow); grade 2: moderately severe lesion with active periosteal proliferation causing enlargement of the DSP dorsally (short arrow) and moderate lysis (long arrow); grade 3: severe osseous pathology showing a severe lytic lesion (long arrow) and periosteal proliferation (short arrow).

### 2.5. Statistical Analysis

Statistical analyses were performed using SPSS 19.0 for Windows (IBM Corporation, Armonk, NY). Descriptive statistics were calculated, and linear regression was used to evaluate the relationship between VAS scores and grade assignments. The ordinal grades were displayed graphically to visualize regional trends in severity of DSP lesions across all horses. The VAS scores were shown to be normally distributed using the Kolmogorov-Smirnov test and were analyzed using multivariate analysis of variance (ANOVA) with post-hoc Bonferroni tests corrected for multiple samples. Age, size, and vertebral level were fixed factors, and horse was a random factor.

## 3. Results

Enthesophytes were frequently present at the attachment sites of the interspinous ligaments (Fig. 2), especially


Fig. 2. Lateral view of two dorsal spinous processes showing grade 1 interspinous enthesophytes with periosteal proliferation and enlargement at the attachment site of the interspinous ligament (arrows).
in the mid-thoracic region and were most often scored as grade 1. Enthesophytes (grades 1-3) were often found on the cranial side of the dorsal tip of the cranial thoracic DSPs from T1 to T5. Signs of impingement were found on both the cranial and caudal aspects of the DSPs throughout the thoracolumbar spine. In addition to lytic and proliferative changes, impingement was sometimes associated with lateral deviation or overriding of the tips of the DSPs. Fusion or ankylosis of adjacent processes occurred infrequently and pseudoarticulations, including the formation of false joints, was also observed (Fig. 3). Lesions at the base (ventral part) of the DSP were found in the cranial thoracic spine from T1-T3 and from T14-L6 (Fig. 4).

Linear regression analysis showed a highly significant linear relationship between VAS scores and grade assignments ( $\mathrm{R}^{2}=0.976$ ). Lesions with severity $\geq$ grade 1 were present in $97 \%$ of horses. At least one moderate-to-severe lesion $\geq$ grade 2 was found in $82 \%$ of horses, and $52 \%$ of horses had one or more grade 3 lesions. There were regional differences in severity with severe (grade 3) lesions being confined to the cranial thoracic region (T2-T3 and T4-T5), the mid to caudal thoracic region (T11-T12 to T18-L1), and at L4 to L5 (Fig. 5).

For the entire group of horses, the VAS scores (Table 1) showed significantly higher values from T12-13 to T16-T17 than at all other vertebral levels. Old horses had overall higher VAS scores than young horses ( $P<.001$ ) with significantly more severe lesions in old horses at three vertebral levels (Table 1). Tall horses had overall significantly higher VAS scores than short horses $(P<.001)$ with significantly more severe lesions in tall horses at 12 spinal levels (Table 1).

## 4. Discussion

The results presented here indicate that osseous lesions occur frequently on the DSPs of the equine thoracolumbar spine. In accordance with our experimental hypothesis, severe lesions were found in the mid to caudal thoracic region, which had been predicted based on the relatively


Fig. 3. Lateral views showing grade 3 impingements of the dorsal spinous processes of adjacent vertebrae. Left: grade 3 lesion with lysis, proliferation, and pseudoarticulation formation. Right: Grade 3 lesion with proliferation, lysis, and flattening of the vertebral surfaces.
large range of motion, combined with the lordotic effect of the rider's weight in an area that has relatively poor mechanical support [ $8,18,21$ ]. The significantly greater severity of lesions in the cranial thoracic region and at L4-L5 was not predicted. The hypotheses that lesion severity would increase with age and height of the horse were both supported, although not at all vertebral levels.

Previous studies of DSP osseous lesions have been based on descriptions of the lesions [3,8] or ordinal grading systems [5,20]. The use of VAS has become common in the medical field, particularly in relation to self-assessed pain scores [22]. The linear relationship between the ordinal grades and the continuous VAS scores reported here $\left(R^{2}=\right.$ 0.976 ) indicates an excellent level of agreement between the two scoring systems. A similarly good level of agreement between ordinal grades and VAS scores was reported in a study of osseous pathology of the SIAs [23]. The advantages of using VAS scores is that they combine observations of a large range of pathologic changes in a single score, they allow a finer level of gradations in the scores, and they are amenable to statistical analysis using ANOVA. The authors recommend the use of VAS scores be considered in future studies of spinal pathology.

These data add to previous studies of vertebral osseous pathologies by evaluating horses of a variety of breeds, types, and sizes and evaluating the entire thoracolumbar spine. The severe DSP osseous lesions found in the mid to caudal thoracic region agree with necropsy findings in Thoroughbred racehorses $[2,19,20$ ] and with clinical findings based on radiology, scintigraphy, or ultrasonography [3,4,9]. Caudal to T12, which lies at the caudal part of the withers, the DSPs are wider [16], and the intervertebral
spaces are correspondingly narrower $[3,17]$ than in the region cranial to T 12 . It is notable in this regard that the DSPs forming the caudal slope of the withers had only a small number of grade 1 lesions and no lesions of grade 2 or 3.

The severe osseous lesions in the first few thoracic vertebrae may reflect the large ranges of motion in flexionextension and axial rotation ex vivo especially at T1 to T2 [24]. The greater mobility in this region may be related to changes in morphology of the intervertebral joints [25] together with the absence of the stabilizing effect of the supraspinous ligament cranial to T3 [15] and the greater thickness of the intervertebral discs at T1-T2 and T2-T3 compared with the more caudal discs [24]. A wide range of motion is necessary at the cervicothoracic junction to allow the neck and head to be moved through a large range of motion in the sagittal [26] and dorsal [27] planes. We hypothesize that high forces are generated in this region when the horse lands from a large fence with the trunk oriented at a high angle relative to the ground and the neck elevated. Ground reaction forces are transmitted through the thoracic synsarcosis to the thoracic vertebral column, which is loaded in compression during landing. Instability of the first few thoracic vertebrae associated with the short DSPs and lack of a supraspinous ligament, may allow micromotion between adjacent vertebrae that increases the likelihood of developing osteoarthritic lesions at sites of impingement. This may be responsible for some of the lytic and proliferative lesions found at the base of the first two thoracic vertebrae.

The thoracolumbar vertebrae are firmly united by soft tissues that include the intervertebral disks, joint capsules,


Fig. 4. Impingement of the ventral part of the dorsal spinous processes (DSPs) in the cranial thoracic region. Left: lateral view of a grade 2 lesion on the caudoventral aspect of the DSP of T1. Right: caudal view of a grade 3 lesion on the caudoventral aspect of the DSP of T2 showing considerable lysis.


Fig. 5. Graph showing the percentage of horses $(\mathrm{n}=33$ ) with grade 0 (light blue), 1 (green), 2 (dark blue), and 3 (black) osseous lesions of the dorsal spinous processes at each intervertebral level from the cervicothoracic junction to the lumbosacral junction.
intervertebral ligaments, and perivertebral muscles with their associated tendons and fascia. These tissues exert sufficient tension that the vertebrae of quadrupeds are loaded in compression in spite of their horizontal

Table 1
Mean $\pm$ standard deviation visual analog scores for osseous lesions of the dorsal spinous processes separated by age (young $<15$ years; old $\geq 15$ years) and height (short $<152 \mathrm{~cm}$; tall $\geq 152 \mathrm{~cm}$ ) and overall group values.

| Level | Age |  | Height |  | Total <br> All Horses |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  | Old | Young | Tall | Short |  |
| C7.T1 | $3.2 \pm 3.0$ | $2.8 \pm 2.0$ | $2.8 \pm 2.8$ | $3.4 \pm 2.2$ | $3.0 \pm 2.5$ |
| T1.2 | $5.9 \pm 4.5$ | $2.9 \pm 3.5$ | $5.7 \pm 4.4$ | $\mathbf{2 . 1} \pm \mathbf{3 . 0}$ | $4.3 \pm 4.3$ |
| T2.3 | $5.3 \pm 3.6$ | $5.1 \pm 7.3$ | $6.2 \pm 5.6$ | $3.7 \pm 5.8$ | $5.2 \pm 5.7$ |
| T3.4 | $6.1 \pm 4.3$ | $\mathbf{2 . 0} \pm 2.5$ | $4.5 \pm 3.8$ | $3.4 \pm 4.3$ | $4.1 \pm 4.0$ |
| T4.5 | $5.6 \pm 3.5$ | $4.3 \pm 3.7$ | $5.8 \pm 3.7$ | $3.7 \pm 3.1$ | $5.0 \pm 3.6$ |
| T5.6 | $5.1 \pm 2.7$ | $4.5 \pm 2.5$ | $5.5 \pm 2.3$ | $3.9 \pm 2.6$ | $4.8 \pm 2.5$ |
| T6.7 | $5.3 \pm 2.0$ | $5.1 \pm 1.8$ | $5.6 \pm 1.5$ | $4.5 \pm 2.2$ | $5.2 \pm 1.8$ |
| T7.8 | $4.7 \pm 2.5$ | $4.4 \pm 2.0$ | $5.1 \pm 1.9$ | $3.7 \pm 2.6$ | $4.5 \pm 2.3$ |
| T8.9 | $4.5 \pm 2.5$ | $3.8 \pm 2.6$ | $5.2 \pm 1.8$ | $2.7 \pm 2.8$ | $4.1 \pm 2.5$ |
| T9.10 | $3.1 \pm 2.3$ | $3.8 \pm 2.9$ | $4.4 \pm 2.4$ | $2.1 \pm 2.4$ | $3.4 \pm 2.6$ |
| T10.11 | $4.6 \pm 2.9$ | $3.6 \pm 3.2$ | $4.7 \pm 2.9$ | $3.1 \pm 3.1$ | $4.1 \pm 3.0$ |
| T11.12 | $5.9 \pm 5.3$ | $6.1 \pm 3.8$ | $7.8 \pm 4.5$ | 3.3. $\pm 3.3$ | $6.0 \pm 4.6$ |
| T12.13 | $7.2 \pm 4.9$ | $6.9 \pm 3.7$ | $8.5 \pm 4.2$ | $4.9 \pm 3.5$ | $7.0 \pm 4.3$ |
| T13.14 | $8.3 \pm 4.1$ | $9.1 \pm 4.4$ | $9.3 \pm 3.7$ | $7.9 \pm 4.8$ | $8.7 \pm 4.2$ |
| T14.15 | $9.5 \pm 5.2$ | $8.1 \pm 4.6$ | $10.2 \pm 4.6$ | $6.7 \pm 4.6$ | $8.8 \pm 4.9$ |
| T15.16 | $10.6 \pm 5.1$ | $8.9 \pm 5.1$ | $11.4 \pm 5.0$ | $7.1 \pm 4.1$ | $9.7 \pm 5.1$ |
| T16.17 | $11.5 \pm 5.1$ | $8.8 \pm 5.3$ | $12.0 \pm 5.1$ | $7.2 \pm 4.4$ | $10.1 \pm 5.3$ |
| T17.18 | $9.3 \pm 6.2$ | $7.1 \pm 6.2$ | $10.3 \pm 6.6$ | $4.6 \pm 3.3$ | $8.1 \pm 6.2$ |
| T18.L1 | $7.9 \pm 5.6$ | $5.0 \pm 4.8$ | $8.3 \pm 5.8$ | $3.5 \pm 3.2$ | $6.4 \pm 5.4$ |
| L1.L2 | $6.7 \pm 3.9$ | $3.9 \pm 3.0$ | $6.5 \pm 3.8$ | $3.3 \pm 2.6$ | $5.3 \pm 3.7$ |
| L2.L3 | $5.4 \pm 3.0$ | $3.8 \pm 2.8$ | $5.4 \pm 2.9$ | $3.3 \pm 2.8$ | $4.6 \pm 3.0$ |
| L3.L4 | $4.0 \pm 2.2$ | $4.3 \pm 2.8$ | $4.1 \pm 2.4$ | $4.2 \pm 2.8$ | $4.2 \pm 2.5$ |
| L4.L5 | $9.5 \pm 5.4$ | $6.5 \pm 4.2$ | $9.5 \pm 5.2$ | $5.6 \pm 3.7$ | $8.0 \pm 5.0$ |
| L5.L6 | $4.1 \pm 4.1$ | $2.4 \pm 2.3$ | $2.9 \pm 3.6$ | $3.7 \pm 2.9$ | $3.2 \pm 3.3$ |
| L5/6.S1 | $2.3 \pm 3.3$ | $0.3 \pm 0.7$ | $1.7 \pm 2.9$ | $0.5 \pm 1.1$ | $1.2 \pm 2.4$ |

Bolded pairs of values differ significantly by age or height as determined by analysis of variance and Bonferroni post hoc testing.
orientation [28]. The thoracolumbar spine is supported cranially by the forelimbs and caudally by the hindlimbs with the considerable weight of the horse's viscera, and sometimes the additional weight of the rider, applying an extensor force to the spine. The lordotic effect is greatest in the middle part of the back, which is furthest from the limb supports, and where there is also some lateral bending and axial rotational movement [24]. This is the region that shows the most severe osseous DSP lesions both in this and other studies [2,19,24].

In gaits that have a suspension phase, sagittal plane oscillations of the thoracolumbar spine occur in rhythm with limb movements. At trot, the intervertebral joints flex and the back rounds during the suspension phase then extend during diagonal stance [29]. The addition of the rider's weight to the middle part of the horse's back contributes to thoracolumbar lordosis both at stance and during locomotion causing the lumbar region to be more extended throughout the stride at walk, trot, and canter although the range of motion in flexion-extension does not change [18].

The interspinous ligaments, which unite adjacent DSPs along the length of the processes, together with the supraspinous ligament that attaches along the dorsal border limit distraction of the DSPs and flexion of the intervertebral joints. The supraspinous ligament, which is the caudal continuation of the nuchal ligament, is composed of dense fibrous tissue. It is strongest in the cranial thoracic region and is thick and dense in the lumbar region but is more elastic in the mid to caudal thoracic region where it fuses with the lumbodorsal fascia and the tendons of insertion of the m . longissimus dorsi [30]. When the intervertebral joints flex, the DSPs tend to be separated, and the resulting tension in the supraspinous and interspinous ligaments may be associated with the development of enthesophytes. Supraspinous ligament desmopathy, which is recognized
clinically and ultrasonographically, is often associated with the development of enthesophytes at the ligamentous attachment sites on the dorsal tip of the DSPs. Injuries of the supraspinous ligament occur most frequently between T15 and L3 [1]. When the intervertebral joints extend, the adjacent DSPs are approximated and may impinge on each other, leading to the development of proliferative or lytic lesions.

Impinging DSPs is the most common osseous spinal pathology of horses [3,5,8,11,12]. It occurs when the interspinous space is too narrow or the horse's posture is excessively lordotic. If the horse is worked in a more rounded posture, it may help to avoid clinical progression in horses with asymptomatic impingement. A radiographic study showed greater separation of the DSPs with the head and neck lowered and closer approximation with the head and neck raised [16]. Bone-on-bone abrasion may result in the development of eburnation, an ivory-like increased bone density, periosteal proliferation, osteophytes, pseudoarthroses and even complete arthrodesis [1,2,19]. It is well recognized that osseous pathology of the DSPs is not necessarily associated with clinical signs [1,11,14] and that a variety of radiological and scintigraphic abnormalities can be present in the DSPs of clinically normal horses [11,13,14]. However, there is a significant association between radiological grades and thoracolumbar pain and between radiological and scintigraphic abnormalities and thoracolumbar pain [31]. The source of pain is not well understood, but inflammation of soft tissues anchored on the spinous processes and nerve endings in the periosteum are likely contributors [3].

Horses with clinically significant impinging DSPs usually exhibit nonspecific signs, such as altered hindlimb gait, resistance to work, toe dragging, poor jumping performance, and rigid posture of the thoracolumbar spine [3,4,32]. Although they do not usually have an adverse reaction to palpation of the DSP [33], affected horses may be painful when ridden and may show persistent bucking, rearing, refusing to jump, and other forms of resistance that may be sufficiently serious to result in their loss of use as riding horses [3,34,35].

In the study reported here, taller horses had more severe DSP osseous lesions at 12 spinal levels. Body mass is closely correlated with height [36], but the strength of the supporting tissues does not increase proportionally with body weight; small horses support larger loads per kilogram body weight than large horses [37]. Consequently, in large horses, the greater extending forces on the thoracolumbar spine are not balanced by a comparable increase in forces provided by the soft tissues to combat overextension, and this may contribute to the greater severity of DSP impingement.

## 5. Conclusions

In conclusion, the results of this study confirm that osseous lesions of the DSPs occur frequently in horses with more severe lesions being found in the cranial thoracic, mid to caudal thoracic, and mid-lumbar regions. The presence of severe osseous DSP lesions in the cranial thoracic and lumbar regions presents a challenge for diagnostic imaging,
so lesions in these areas may be underdiagnosed. The anatomy and mechanics suggest that the signs are likely to be exacerbated by ridden exercise.

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## References

[1] Denoix JM, Dyson S. Thoracolumbar spine. In: Ross MW, Dyson SJ, editors. Diagnosis and management of lameness in the horse. 2nd ed. Philadelphia: W.B Saunders Co; 2009. p. 592-605.
[2] Haussler KK. Osseous spinal pathology. Vet Clin North Am Equine Pract 1999;15:103-12.
[3] Jeffcott LB. Disorders of the thoracolumbar spine of the horse-a survey of 443 cases. Equine Vet J 1980;12:197-210.
[4] Girodroux M, Dyson S, Murray R. Osteoarthritis of the thoracolumbar synovial intervertebral articulations: clinical and radiographic features in 77 horses with poor performance and back pain. Equine Vet J 2009;41:130-8.
[5] Haussler KK, Stover SM, Willits NH. Pathologic changes in the lumbosacral vertebrae and pelvis in Thoroughbred racehorses. Am J Vet Res 1999;60:143-53.
[6] Ranner W, Gerhards H. Vorkommen und Bedeutung von Rückenerkrankungen - inbesondere des "Kissing Spine"-Syndroms - bei Pferden in Süddeutschland. Pferdeheilkunde 2002;18:21-3.
[7] Stubbs NC, Hodges PW, Jeffcott LB, Cowin G, Hodgson DR, et al Functional anatomy of the caudal thoracolumbar and lumbosacral spine in the horse. Equine Vet J 2006;36(Suppl 36):393-9.
[8] Townsend HG, Leach DH, Doige CE, Kirkaldy-Willis WH. Relationship between spinal biomechanics and pathological changes in the equine thoracolumbar spine. Equine Vet J 1986;18:107-12.
[9] Walmsley J, Pettersson H, Winberg F, McEvoy F. Impingement of the dorsal spinous processes in two hundred and fifteen horses: case selection, surgical technique and results. Equine Vet J 2002;34:23-8
[10] Zimmerman M, Dyson S, Murray R. Comparison of radiographic and scintigraphic findings of the spinous processes in the equine thoracolumbar region. Vet Radiol Ultrasound 2011;52:661-71.
[11] Jeffcott L. Radiographic features of the normal equine thoracolumbar spine. Vet Radiol 1979;20:140-7.
[12] Cousty M, Retureau C, Tricaud C, Geffroy O, Caure S. Location of radiological lesions of the thoracolumbar column in French trotters with and without clinical signs of back pain. Vet Rec 2010;166:41-5.
[13] Erichsen C, Eksell P, Roethlisberger Holm K, Lord P, Johnston C. Relationship between scintigraphic and radiographic evaluations of spinous processes in the thoracolumbar spine in riding horses without clinical signs of back problems. Equine Vet J 2004;36:45865.
[14] Holmer M, Wallanke B, Stadtbäumer G. Roëntgenveränderungen an den Dornfortsätzen von 295 klinisch rückgengesunden Warmblutpferden. Pferdeheilkunde 2007;23:507-11.
[15] Nickel R, Schummer A, Seiferle E. The anatomy of the domestic animals, Volume 1: the locomotor system of the domestic mammals. New York: Springer-Verlag; 2006. p. 46-8. 178.
[16] Berner D, Winter K, Brehm W, Gerlach K. Influence of head and neck position on radiographic measurement of intervertebral distances between thoracic dorsal spinous processes in clinically sound horses. Equine Vet J 2012;44(Suppl 43):21-6.
[17] Sinding MF, Berg LC. Distances between thoracic spinous processes in Warmblood foals: a radiographic study. Equine Vet J 2010;42 500-3.
[18] De Cocq P, van Weeren PR, Back W. Effects of girth, saddle and weight on movements of the horse. Equine Vet J 2004;36:758-63.
[19] Stubbs NC. Epaxial musculature and its relationship with back pain in the horse. PhD Thesis, School of Agriculture and Food Sciences, The University of Queensland 2012.
[20] Stubbs NC, Riggs CM, Clayton HM, Hodges PW, Jeffcott LB, et al. Osseous spinal pathology and epaxial muscle ultrasonography in Thoroughbred racehorses. Equine Vet J 2010;42(Suppl. 38):654-61
[21] Audigié F, Pourcelot P, Degueurce C, Denoix JM. Kinematics of the equine back: flexion-extension movements in sound trotting horses. Equine Vet J 1999;31:210-3.
[22] Gift AG. Visual analog scales: measurement of subjective phenomena. Nurs Res 1989;38:286-8.
[23] VanderBroek A, Stubbs NC, Clayton HM. Osseous lesions of the synovial intervertebral articulations in the equine thoracolumbar spine. J Equine Vet Sci 2016;44:67-73.
[24] Townsend HG, Leach DH, Fretz PB. Kinematics of the equine thoracolumbar spine. Equine Vet J 1983;15:117-22.
[25] Townsend HG, Leach DH. Relationship between intervertebral joint morphology and mobility in the equine thoracolumbar spine. Equine Vet J 1984;16:461-5.
[26] Clayton HM, Kaiser LJ, Lavagnino M, Stubbs NC. Dynamic mobilisations in cervical flexion: effects on intervertebral angulations. Equine Vet J Suppl 2010:688-94.
[27] Clayton HM, Kaiser LJ, Lavagnino M, Stubbs NC. Evaluation of intersegmental vertebral motion during performance of dynamic mobilization exercises in cervical lateral bending in horses. Am J Vet Res 2012;73:1153-9.
[28] Alini M, Eisenstein SM, Ito K, Little C, Kettler AA, et al. Are animal models useful for studying human disk disorders/degeneration? Eur Spine J 2008;17:2-19.
[29] Robert C, Valette JP, Pourcelot P, Audigié F, Denoix JM. Effects of trotting speed on muscle activity and kinematics in saddlehorses. Equine Vet J Suppl 2002:295-301.
[30] Jeffcott LB, Dalin G. Natural rigidity of the horse's backbone. Equine Vet J 1980;12:101-8.
[31] Weaver MP, Jeffcott LB, Nowak M. Back problems. Radiology and scintigraphy. Vet Clin North Am Equine Pract 1999;15:113-29.
[32] Gillen A, Dyson S, Murray R. Nuclear scintigraphic assessment of the thoracolumbar synovial intervertebral articulations. Equine Vet J 2009;41:534-40.
[33] Cauvin E. Assessment of back pain in horses. Vet Clin North Am Equine Pract 1997;19:522-33.
[34] Penell J, Egenvall A, Bonnett BN, Olsen P, Pringle J. Specific causes of morbidity among Swedish horses insured for veterinary care between 1997 and 2000. Vet Rec 2005;157:470-7.
[35] Sardari K. Back pain: a significant cause of poor performance in show jumping horses (diagnostic challenge and treatment). Iran J Vet Surg 2008;2:163-9.
[36] Clayton HM, Buchholz R, Nauwelaerts S. Relationship between morphological and stabilographic variables in standing horses. Vet J 2013;198:e65-9.
[37] Van den Bogert AJJ, Meershoek L, Hof A. Mechanical analysis and scaling. In: Back W, Clayton HM, editors. Equine locomotion. 2nd ed. New York: Saunders Elsevier; 2013. p. 443-65.


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