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# Epaxial muscle atrophy is more evident in large dogs with intervertebral disc disease than in dogs with ischaemic myelopathy

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ABSTRACT

Cross-sectional area (CSA) decreases and fat infiltration increases in epaxial muscles of Dachshunds with intervertebral disc disease (IVDD), but less is known about large breed dogs with IVDD. The aim here was to investigate thoracolumbar epaxial muscle CSA and fat infiltration in large breed dogs with compressive IVDD and acute non-compressive nucleus pulposus extrusion (ANNPE) or fibrocartilaginous embolism (FCE).

This retrospective study included large breed dogs with MRI-confirmed IVDD ($n = 17$) and ANNPE or FCE ($n = 13$). The CSA and fat infiltration of the thoracolumbar *M. longissimus* and Mm. multifidi were assessed from T1-weighted transverse MR images using Osirix.

The CSA was significantly smaller in dogs with compressive IVDD than in dogs with non-compressive ANNPE or FCE for Mm. multifidi ($p = 0.015$), *M. longissimus* ($p = 0.070$), and these two muscles combined ($p = 0.016$). Fat infiltration in all muscle measurements was significantly higher in dogs with compressive IVDD than in dogs with non-compressive ANNPE or FCE (all $P < 0.050$). A significant positive correlation existed between age, duration of clinical signs, and fat infiltration, suggesting more fat infiltration in older dogs with more chronic signs.

These signs of muscle atrophy are likely caused by denervation and secondary disuse due to chronic spinal cord compression and prolonged duration of clinical signs.

## 1. Introduction

Investigation of epaxial muscle size and composition in relation to intervertebral disc disease (IVDD) and lumbosacral stenosis is currently a growing research interest in veterinary medicine (Boström et al., 2014; Cain et al., 2016; Henderson et al., 2015; Lerer et al., 2015). Human research has found decreased cross-sectional area (CSA) and increased fat infiltration in the spinal muscles, particularly in the Mm. multifidi, on magnetic resonance imaging (MRI) and computed tomography (CT) in people with back pain (D'Hooge et al., 2012; Goubert et al., 2016; Goubert et al., 2017). These degenerative changes in the muscles alter the contractile ability of the affected muscle, resulting in compromised muscle function (D'Hooge et al., 2012). The degenerative changes and compromised function in the muscles may persist although the instigating injury heals and the pain resolves (Battié et al., 2012; Hides et al., 1996).

In humans, Mm. multifidi and M. erector spinae are considered important muscles for dynamic stabilization of the spine, and back problems in humans are managed with specific, targeted exercise and general physical activity with the aim of restoring neuromuscular function in the spinal muscles (Macedo et al., 2012; Macdonald et al., 2006; Tsao et al., 2010; Richards et al., 2013). Such management is not yet commonly used in veterinary medicine, although anatomy and function of the human and canine spinal musculature are similar (Evans,

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1993; Ritter et al., 2001).

In dogs with spinal pathology, the epaxial muscles show signs of atrophy, similar to those found in humans (Boström et al., 2014; Lerer et al., 2015). A recent study reported that Dachshunds with compressive IVDD had significantly smaller CSA and greater fat infiltration in the Mm. multifidi and *M. longissimus* than dogs with FCE (Boström et al., 2014). Contrary to this, another retrospective MRI study on 180 patients found higher fat infiltration in Mm. multifidi and M. psoas in non-chondrodystrophic dogs than in chondrodystrophic dogs (Lerer et al., 2015). They also noted that fat infiltration was higher in dogs with non-IVDD spinal pathology than in dogs with IVDD (Lerer et al., 2015). Moreover, larger dogs with lumbosacral stenosis have decreased CSA in their lumbosacral epaxial muscles compared with healthy controls (Cain et al., 2016; Henderson et al., 2015), and researchers suggest that the dynamic stabilization of the spine is compromised due to these degenerative changes in the epaxial muscles (Cain et al., 2016; Henderson et al., 2015). To date, no studies have investigated the epaxial muscle size and composition in large breed dogs with thoracolumbar IVDD.

Hansen type II IVDD usually affects older large breed dogs (Hansen 1951; Macias et al., 2002). The onset of clinical signs is chronic progressive as the annulus fibrosus weakens, causing nucleus pulposus to protrude dorsally into the vertebral canal, resulting in gradual spinal cord compression and often slowly progressive neurological deficits (Crawford and De Decker, 2017; Macias et al., 2002). Sometimes acute extrusions occur in addition to the chronic protrusions, causing acute exacerbation of the chronic neurological deficits (Crawford and De Decker, 2017). Treatment can be surgical or medical (Fenn et al., 2016) and physiotherapy may be prescribed (Olby et al., 2005; Sims et al., 2015), but recovery may be difficult due to chronic pathological changes in the spinal cord (Crawford and De Decker, 2017; Macias et al., 2002).

Ischaemic myelopathy (FCE) is related to fibrocartilaginous material embolizing a spinal cord blood vessel, causing ischaemia and resulting in local spinal cord necrosis (Fenn et al., 2016; Gandini et al., 2003; De Risio et al., 2008). Acute non-compressive nucleus pulposus extrusion (ANNPE) causes contusion of the spinal cord instead of ischaemic injury by rapid extrusion of non-degenerative nucleus pulposus material (De Risio et al., 2009; Fenn et al., 2016). Both FCE and ANNPE present with similar clinical signs, including acute, non-progressive, often asymmetrical neurological deficits with no or only mild spinal pain, contrary to most dogs suffering from acute compressive IVDD (Fenn et al., 2016; De Risio et al., 2008, 2009; Gandini et al., 2003).

Supportive treatments and interventions are needed to enhance and maintain muscle function and to prevent recurrence of pain episodes in dogs with compressive IVDD. Knowledge about epaxial muscle size and composition in relation to this disease would provide justification for introducing therapeutic training strategies to treat these dogs. Therefore, the aim of this study was to investigate the thoracolumbar Mm. multifidi and *M. longissimus* muscle size and composition in large breed dogs suffering from compressive spinal cord injury (IVDD) and in dogs with non-compressive spinal cord injury (ANNPE or FCE). We hypothesized that the Mm. multifidi and *M. longissimus* dorsi CSA would be decreased and their fat infiltration increased in dogs with compressive IVDD relative to dogs with non-compressive ANNPE or FCE.

## 2. Materials and methods

### 2.1. Subjects

The clinical database of the Queen Mother Hospital, Royal Veterinary College, University of London was searched retrospectively for large breed dogs with MRI-confirmed thoracolumbar IVDD, FCE, or ANNPE. Inclusion criteria were body weight above 15 kg, complete medical records including duration of clinical signs, neurological examination, and the respective radiological diagnosis. Patients were excluded if MRI scans were not available for review or did not include transverse T1-weighted images of the thoracic or lumbar spine or if an additional diagnosis of degenerative myelopathy, discospondylitis, myelitis, or spinal tumour was made.

The age, breed, sex, and body weight of dogs, diagnosis, site of lesion, duration of clinical signs, and neurological grade at presentation were retrieved from patient records. The duration of clinical signs was categorized according to Boström et al. (2014) as "acute" (=less than 7 days) or "chronic" (=7 days or more). The neurological grade at the time of presentation was determined retrospectively with a modified Frankel score as in Van Wie et al. (2013) as follows: 1 = pain only, 2 = ambulatory paraparesis, 3 = non-ambulatory paraparesis, 4 = paraplegia, and 5 = paraplegia without deep pain perception. Dogs were divided into two groups based on the preliminary diagnosis stated in the medical records: 1 = dogs with compressive spinal cord injury (Hansen type II IVDD, chronic or acute on chronic IVDD) and 2 = dogs with non-compressive spinal cord injury (ANNPE) and (FCE).

### 2.2. Methodology

Magnetic resonance images from a 1.5 Tesla scanner (Phillips Intera, Phillips Medical Systems, Eindhoven, The Netherlands) were reviewed. The dogs were all scanned in dorsal recumbency providing T1-weighted sagittal and transverse images with an image size between 288 × 288 and 400 × 400, echo time TE 8–11 ms, repeat time TR 484–1151 ms, slice thickness 3–3.50 mm, and spacing between slices 3.3–3.85 mm in the transverse plane. The acquisitions were performed with the dogs under general anaesthesia. The anaesthesia protocol varied according to individual patient requirements. All available thoracolumbar segments in each dog were analysed from T1 transverse sequences with Osirix Dicom viewer (Pixmeo, Bernex, Switzerland).

One assessor (BP) was trained in the measurement technique and conducted the measurements in random order, blinded to the diagnosis and background data of all dogs. The individual spinal segments were identified from the sagittal T1 sequence. A previously reported and reliable manual drawing technique was used to measure the CSA (Boström et al., 2014, 2018; Elliott et al., 2006). A region of interest (ROI) was drawn around the muscle, tracing the muscle borders evident in the transverse T1 images (Fig. 1). Measurements were taken bilaterally from the Mm. multifidi muscles (MM), the combined *M. longissimus* dorsi and M. iliocostalis muscles (EPAX), and the combined Mm. multifidi, *M. longissimus*, and M. iliocostalis muscles (MMEPAX). The Mm. multifidi were measured individually, whereas the *M. longissimus* dorsi and M. iliocostalis were measured together, forming the epaxial muscle measurement, as it was difficult to distinguish a clear anatomical border between these muscles. The muscle measurements were made at the site of the intervertebral discs in the T9–L4 range, and every available transverse slice per intervertebral disc was measured (Fig. 2).

The CSA of the disc (DISC CSA) was measured in the same transverse images as the muscle measurements (Fig. 2 A–C). Discrepancy in body weight and body conformation between the two groups of dogs was accounted for by calculating a muscle-to-disc ratio (Boström et al., 2014):

Muscle:Disc ratio = Muscle CSA / Disc CSA

To provide another standardization method, the vertebral body (VB) CSA was measured at the caudal endplate of the 13th thoracic vertebra in all dogs according to Henderson et al. (2015) and Cain et al. (2016). The following ratio was generated:

Muscle:VB ratio = Muscle CSA / VB CSA

These calculations generated the following Muscle:Disc variables: Mm. multifidi (MM:DISC, MM:VB), *M. longissimus* and M. iliocostalis combined (EPAX:DISC, EPAX:VB), and Mm. multifidi, *M. longissimus*, and M. iliocostalis combined (MMEPAX:DISC, MMEPAX:VB).

Fat infiltration in the muscles was estimated based on the signal intensity in each individual muscle ROI. To allow comparison between subjects, the MRI acquisition variability was accounted for by calculating a signal intensity ratio between the muscle and subcutaneous fat. A standard 0.200 $cm^2$ area of subcutaneous fat was measured at the

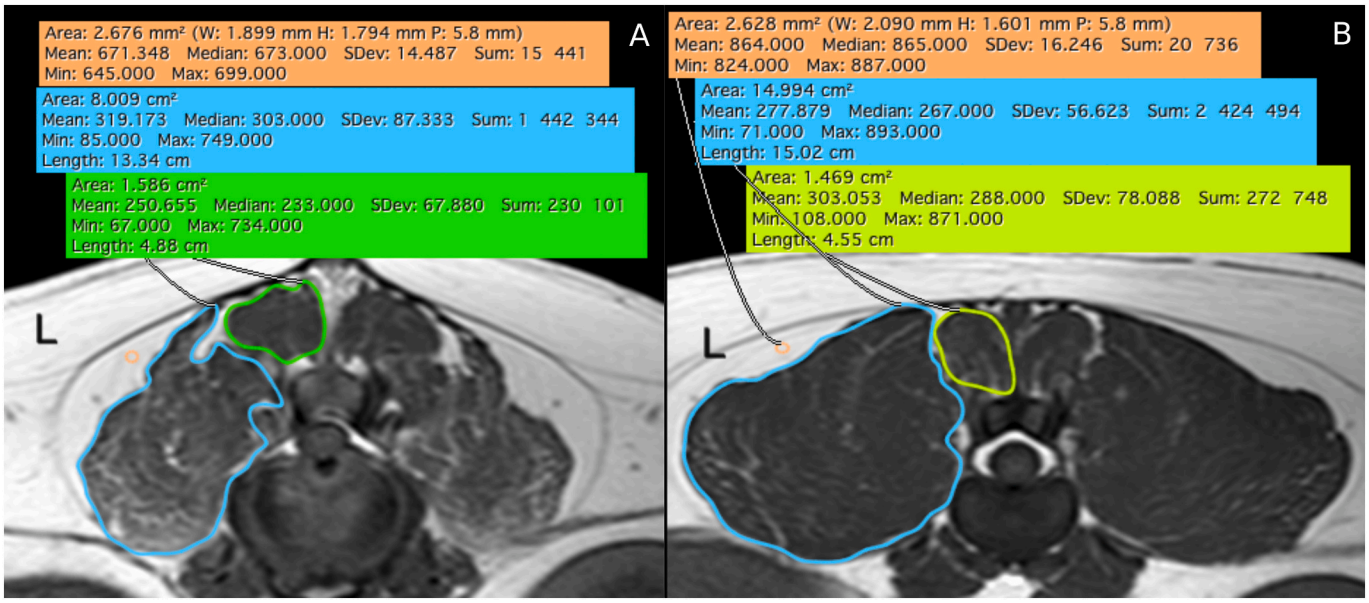


**Fig. 1.** Muscle and subcutaneous fat measurements (CSA in coloured draw areas and ROI details in coloured boxes) on T1 transverse MR images at L1–2 intervertebral disc in a dog with IVDD (A) and a dog with ANNPE/FCE (B). These dogs were of the same breed and similar body weight.

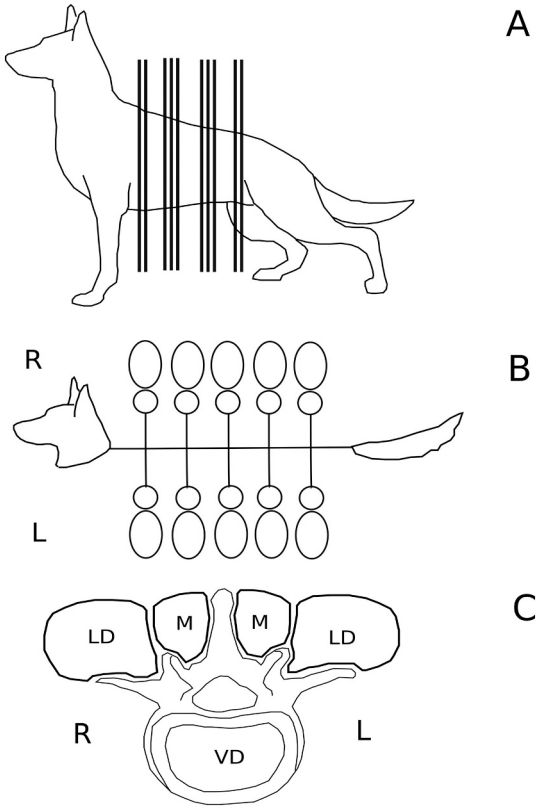


**Fig. 2.** Protocol for measurements. First, each spinal segment (line clusters) in T9-L4 was identified, and all available transverse slices (individual black lines) in each segment were measured (A). B) The measurements from Mm. multifidi (small circles) and *M. longissimus* dorsi (large oval) were obtained from the left and right sides, and an average between the left and right sides was calculated. C) This figure illustrates the CSA measurements of Mm. multifidi (M), *M. longissimus* (LD), and the intervertebral disc (VD) at L2–3 segment.

visually whitest spot in the same image as the muscle measurements. The muscle-to-fat ratio (Muscle:Fat) was calculated as follows (Boström et al., 2014):

Muscle:Fat ratio = Muscle signal intensity mean / subcutaneous fat signal intensity mean

The Muscle:Fat ratio was calculated for all muscle measurements in all dogs providing the following Muscle:Fat variables: Mm. multifidi (MM:FAT), *M. longissimus* and M. iliocostalis combined (EPAX:FAT), and Mm. multifidi, *M. longissimus*, and M. iliocostalis combined (MMEPAX:FAT).

### 2.3. Statistical analysis

The sample size calculation with a confidence level of 95% and a power of 0.8 was conducted based on a previous study investigating transverse area ratios of lumbosacral muscles in dogs with lumbosacral stenosis (Henderson et al., 2015). The transverse area ratio for the lumbar Mm. multifidi was used in the calculation and a sample of 17 dogs in each group was considered sufficient. Descriptive statistics mean and standard deviation (SD) were used to summarize the background data of the dogs. The differences in body weight, age, duration of clinical signs, neurological grade, and disc CSA between the two groups were assessed with Student's *t*-test (continuous variables) and the Mann–Whitney test (non-continuous variables). The mean for the Muscle:Disc ratio, Muscle:VB ratio, and Muscle:Fat ratio from the measured slices was calculated for each muscle (Fig. 2). An average value was calculated from the left and right sides, and these average variables were used in the analysis between groups. Data were tested for normality using the Shapiro-Wilk test and analysed for homogeneity of variances using the Levene test. The Mann–Whitney test was chosen for the comparison between the two groups. The comparisons were made for all measured intervertebral discs and then for only affected vertebral discs.

A general linear model was used to test the possible effects of age, body weight, duration of clinical signs, and neurological grade on the muscle variables. The muscle variables were placed in the model as dependent variables, with the compressive and non-compressive category as a fixed factor and age, body weight, duration of clinical signs,

and neurological grade as covariates. The analysis was conducted separately for all measured intervertebral discs and for only affected intervertebral discs.

In addition, the correlations between the muscle variables and age, duration of clinical signs, and neurological grade were assessed with Spearman correlation coefficient.

Level of significance was set at $P < 0.05$, and the Graph Pad Prism version 5.04 and SPSS, IBM version 24 were used for analysis.

## 3. Results

### 3.1. Descriptive statistics of the studied dogs

Thirty dogs matched the inclusion criteria. The neuroanatomical localization was T3-L3 myelopathy for all dogs. The dogs in the compressive group ($n = 17$) were of the following breeds: German shepherd ($n = 5$), Staffordshire bull terrier ($n = 5$), English pointer ($n = 1$), Golden retriever ($n = 1$), Irish terrier ($n = 1$), Labrador ($n = 1$), Pointer ($n = 1$), Rottweiler ($n = 1$), and mixed breed ($n = 1$). The mean age of the dogs was 8.3 ± 2.4 years and the mean weight 31.1 ± 11.8 kg.

The onset of clinical signs was chronic ($n = 10$) or acute ($n = 7$). The neurological grade at presentation was 2: ambulatory paraparesis ($n = 15$) or 4: paraplegia ($n = 2$).

The diagnosis was intervertebral disc extrusion with and without haemorrhage ($n = 7$), a combination of intervertebral disc extrusion and protrusion ($n = 8$), and intervertebral disc protrusion (n = 2). Eleven dogs had multiple lesion sites. Fifteen of these dogs underwent decompressive surgery via hemilaminectomy and 2 dogs received medical management.

The dogs in the non-compressive group ($n = 13$) comprised a Bullmastiff ($n = 1$), Collie ($n = 1$), German shepherd ($n = 1$), Siberian husky ($n = 1$), Labrador ($n = 4$), Labradoodle ($n = 1$), Lurcher ($n = 1$), Staffordshire bull terrier ($n = 2$), and Welsh sheepdog ($n = 1$). The mean age of the dogs was 6.1 ± 3.2 years and their mean weight 29.0 ± 9.3 kg.

The onset of clinical signs was acute in all dogs ($n = 13$). The neurological grade at presentation was 2: ambulatory paraparesis ($n = 2$), 3: non-ambulatory paraparesis ($n = 7$), 4: paraplegia ($n = 2$), and monoparesis on right side ($n = 2$).

The diagnosis for the dogs in the non-compressive group was ANNPE ($n = 8$) or FCE ($n = 3$). Two dogs had, in addition to the preliminary acute diagnosis of ANNPE or FCE, secondary lesion sites in the form of a chronic disc protrusion. All dogs in this group had medical treatment.

### 3.2. Difference between groups

The dogs in the compressive group were significantly older (8.2 ± 2.4 years vs. 6.1 ± 3.2 years, $P = 0.045$) and had longer duration of clinical signs (63.6 ± 85.5 days vs. 1.2 ± 0.5 days, $P < 0.001$) than the dogs in the non-compressive group. There was no significant difference in body weight or neurological grade between the two groups. The dogs in the compressive group had significantly larger disc CSA (3.5 ± 1.3 $cm^2$) than those in the non-compressive group (2.3 ± 0.8 $cm^2$, $P < 0.0001$). No difference was present in vertebral body CSA between the two groups (compressive 2.1 ± 0.5 $cm^2$ and non-compressive 1.7 ± 0.5 $cm^2$, $P = 0.093$).

In the analysis of all measured intervertebral discs, dogs with compressive IVDD had significantly lower MM:DISC ratio ($P = 0.015$), EPAX:DISC ratio ($P = 0.007$), and MMEPAX:DISC ratio ($P$ 0.015) than dogs with non-compressive ANNPE or FCE (Table 1, Figs. 3–5). The EPAX:VB ratio ($P$ 0.015) and the MMEPAX:VB ratio ($P = 0.017$) were lower in dogs with compressive IVDD than in dogs with non-compressive lesions, but the MM:VB ratio was not significantly different between groups ($P = 0.072$). The MM:FAT ratio, the EPAX:FAT ratio, and the MMEPAX:FAT ratio were significantly higher in the compressive group than in the non-compressive group (all $P < 0.05$) (Table 1, Figs. 3–5).

**Table 1**
Differences in Muscle:Disc, Muscle:VB, and Muscle:Fat ratios between study groups, all intervertebral discs.

| Muscle ratio | C ($n = 17$) | | NC ($n = 13$) | | |
|---|---|---|---|---|---|
| | Mean | *SD* | Mean | *SD* | p |
| EPAX:DISC | 3.498 | *1.796* | 4.792 | *1.358* | 0.007* |
| MM:DISC | 0.479 | *0.245* | 0.605 | *0.239* | 0.015* |
| MMEPAX:DISC | 4.153 | *2.115* | 5.512 | *1.607* | 0.015* |
| EPAX:VB | 4.898 | *1.992* | 6.468 | *1.943* | 0.015* |
| MM:VB | 0.667 | *0.294* | 0.823 | *0.382* | 0.072 |
| MMEPAX:VB | 5.800 | *2.346* | 7.405 | *2.341* | 0.017* |
| EPAX:FAT | 0.346 | *0.053* | 0.297 | *0.036* | 0.008* |
| MM:FAT | 0.346 | *0.055* | 0.302 | *0.047* | 0.020* |
| MMEAX:FAT | 0.350 | *0.052* | 0.300 | *0.038* | 0.002* |

Mean, standard deviation (SD), and significant $p$-value for muscle measurements. All measured sites are included and level of significance was set at $p < 0.05$. C = compressive (IVDD), NC = non-compressive (ANNPE/FCE) spinal cord injury.

In the analysis of only affected vertebral discs, the EPAX:DISC ratio ($P = 0.023$), the MM:VB ratio ($P = 0.013$), the EPAX:VB ratio ($P = 0.039$), and the MMEPAX:VB ratio ($P = 0.001$) were lower in the compressive group than in the non-compressive group (Table 2, Figs. 3–5). The MM:FAT ratio, the EPAX:FAT ratio, and the MMEPAX:FAT ratio were all significantly higher in the compressive group (Table 2) than in the non-compressive group (all $P < 0.050$). There was no significant effect of any of the covariates on the muscle variables.

Considering all measured intervertebral discs and only affected intervertebral discs, there was mild negative correlation between age and the Muscle:Disc ratio for all muscles ($P < 0.050$). Of the Muscle:VB ratio variables, only MMEPAX:VB ratio correlated negatively with age ($P < 0.0001$). There was mild positive correlation between age and the Muscle:fat ratio for all muscles ($P < 0.050$) (Table 3). There was mild negative correlation between duration of clinical signs and both the Muscle:Disc ratio and the Muscle:VB ratio for all muscles ($P < 0.050$), except Mm. multifidi. There was positive correlation between duration of clinical signs and the Muscle:fat ratio for all muscles ($P < 0.050$) (Table 3). No correlation was found between neurological grade and the muscle variables.

## 4. Discussion

This study found smaller CSA and higher fat infiltration in the Mm. multifidi and *M. longissimus* dorsi muscles in large breed dogs with compressive IVDD than in dogs with ANNPE or FCE. The fat infiltration correlated with age and duration of clinical signs such that older dogs with chronic lesions and longer lasting clinical signs had more fat infiltration in their muscles.

We hypothesized that Mm. multifidi and *M. longissimus* dorsi CSA would be decreased in dogs with compressive IVDD relative to dogs with non-compressive ANNPE or FCE lesions. Considering both the analyses of all intervertebral discs and only affected discs, dogs with compressive IVDD had a significantly lower Muscle:Disc ratio and Muscle:VB ratio for most of the muscle measurements. This suggests smaller muscles in the compressive IVDD dogs than in dogs with non-compressive ANNPE or FCE. These results agree with previous studies that reported decreased CSA in the epaxial muscles of Dachshunds with compressive IVDD compared with small breed dogs with FCE (Boström et al., 2014) and decreased CSA in the lumbosacral epaxial muscles of Belgian and German shepherds with lumbosacral stenosis compared with individuals without this disorder (Cain et al., 2016; Henderson et al., 2015). There was also a mild negative correlation between duration of clinical signs and the Muscle:Disc ratio and Muscle:VB ratio for most muscle measurements, indicating smaller muscles in dogs with a longer duration of clinical signs. The decreased muscle CSA here may be due to spinal cord injury causing denervation to the muscles or general disuse atrophy

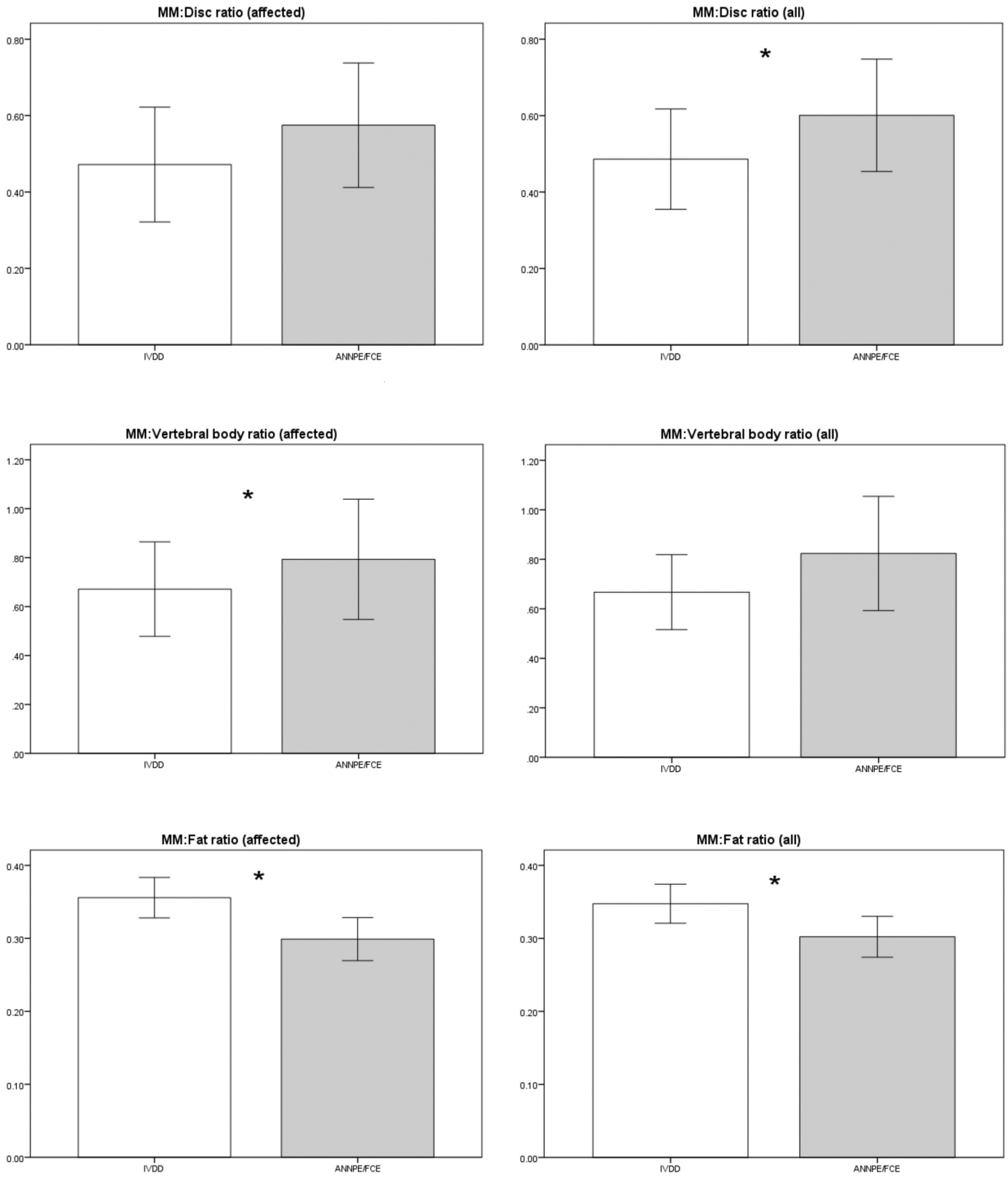


**Fig. 3.** Bar charts showing the statistical significance between the compressive (IVDD) and non-compressive (ANNPE/FCE) groups for Mm. multifidi muscle measurements (MM:Disc ratio, MM:Vertebral body ratio, and MM:Fat ratio) when all and only affected intervertebral discs were included in the analysis. The asterisk indicates statistical significance ($p < 0.050$). MM = Mm. multifidi.

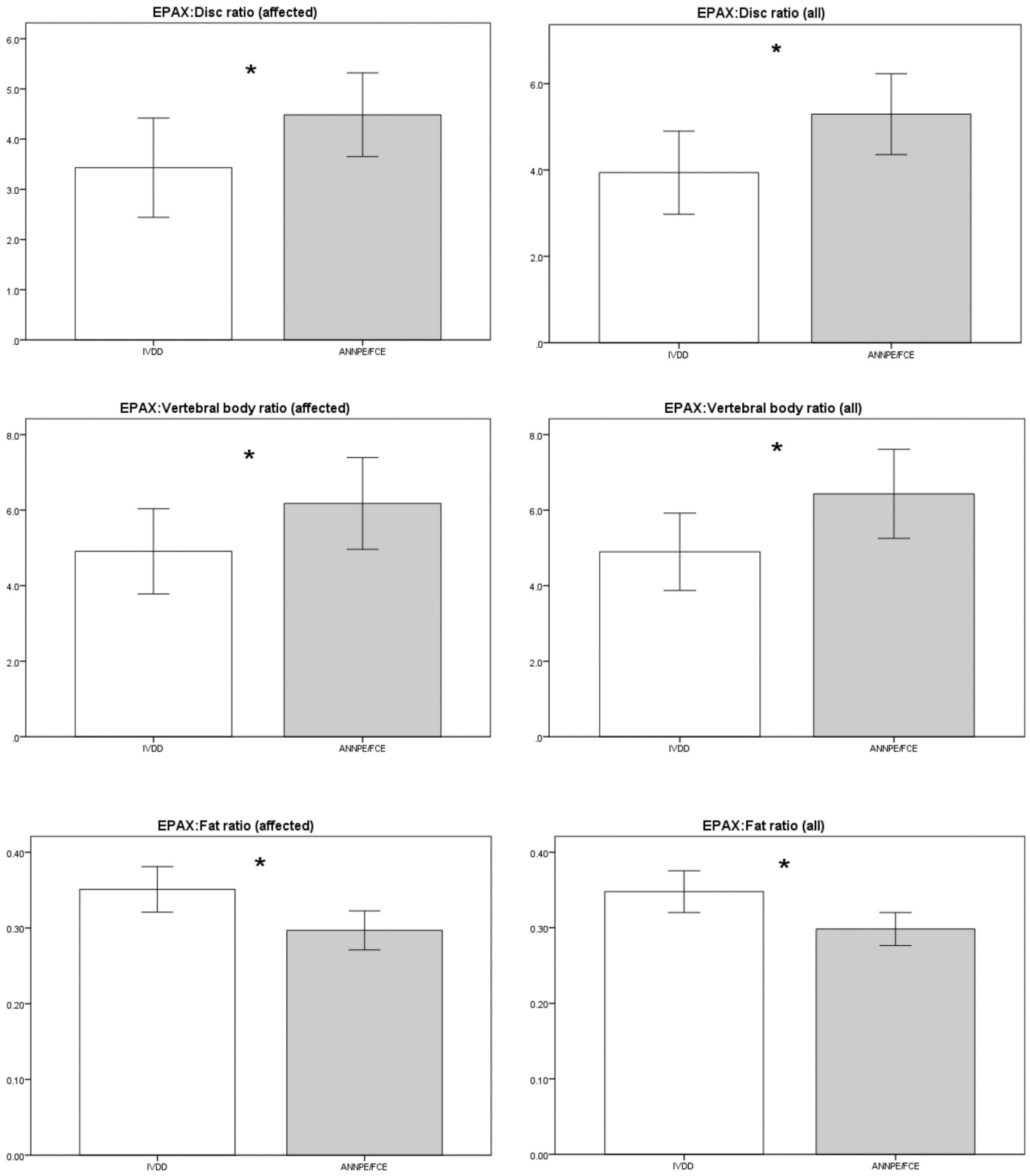


**Fig. 4.** Bar charts showing the statistical significance between the compressive (IVDD) and non-compressive (ANNPE/FCE) groups for *M. longissimus* and M. iliocostalis muscle measurements (EPAX:Disc ratio, EPAX:Vertebral body ratio, and EPAX:Fat ratio) when all and only affected intervertebral discs were included in the analysis. The asterisk indicates statistical significance ($p < 0.050$). EPAX = *M. longissimus* dorsi and M. iliocostalis.

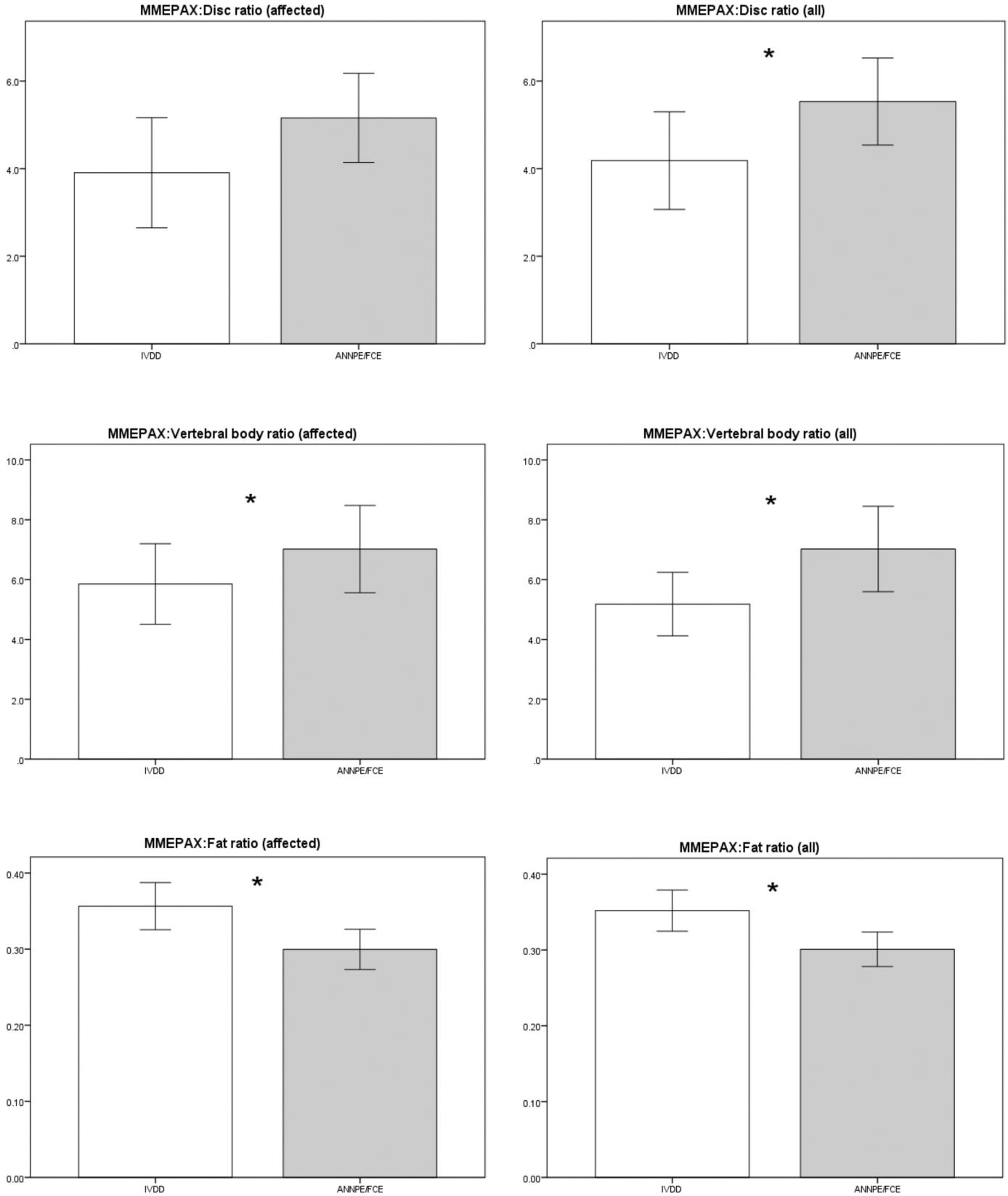


**Fig. 5.** Bar charts showing the statistical significance between the compressive (IVDD) and non-compressive (ANNPE/FCE) groups for *M. longissimus* muscle measurements (MMEPAX:Disc ratio, MMEPAX:Vertebral body ratio, and MMEPAX:Fat ratio) when all and only affected intervertebral discs were included in the analysis. The asterisk indicates statistical significance ($p < 0.050$). MMEPAX = *M. longissimus* dorsi and M. iliocostalis and Mm. multifidi combined.

**Table 2**
Difference in Muscle:Disc, Muscle:VB, and Muscle:Fat ratios between study groups, only affected intervertebral discs.

| Muscle | C (n = 17) | | NC (n = 13) | | |
|---|---|---|---|---|---|
| | Mean | *SD* | Mean | *SD* | p |
| EPAX:DISC | 3.432 | *1.922* | 4.486 | *1.325* | 0.023* |
| MM:DISC | 0.472 | *0.292* | 0.575 | *0.260* | 0.092 |
| MMEPAX:DISC | 4.084 | *2.277* | 5.159 | *1.617* | 0.053 |
| EPAX:VB | 4.610 | *1.748* | 6.179 | *2.010* | 0.039* |
| MM:VB | 0.643 | *0.341* | 0.793 | *0.406* | 0.013* |
| MMEPAX:VB | 5.845 | *2.642* | 7.095 | *2.440* | 0.001* |
| EPAX:FAT | 0.351 | *0.058* | 0.296 | *0.040* | 0.007* |
| MM:FAT | 0.356 | *0.054* | 0.299 | *0.047* | 0.005* |
| MMEPAX:FAT | 0.356 | *0.060* | 0.300 | *0.042* | 0.010* |

Mean, standard deviation (SD), and significant *P*-value for muscle measurements. Only affected sites were included in the analysis and the level of significance was set at $p < 0.05$. C = compressive (IVDD), NC = non-compressive (ANNPE/FCE) spinal cord injury.

secondary to the prolonged, but mild spinal cord compression. In humans, disuse atrophy is already detectable within two weeks following immobilization (Wall et al., 2014), however, decreased muscle mass in older individuals is also considered a normal component of the ageing process (Narici and Maffulli, 2010). The dogs in our compressive IVDD group were significantly older than the dogs in the non-compressive group. Therefore, the identified muscle atrophy could also occur as a normal response to ageing or because older dogs are generally less active than younger ones.

The larger disc CSA in the compressive group may have influenced the Muscle:Disc ratio such that the ratio was smaller in the compressive group. Previous studies have found no difference in the disc area between IVDD Dachshunds and FCE dogs (Boström et al., 2014). However, contrary to the acute disc extrusion reported in IVDD Dachshunds (Boström et al., 2014), the dogs in this study had more chronic and multifocal lesions. It may be that the chronic disorganization of the annulus fibrosus has altered the disc CSA on MRI (Bergknut et al., 2013; Smolders et al., 2013), making it appear larger, but it is also possible that the degeneration of the annulus fibrosus results in the enlarged disc CSA. When comparing muscle measurements, it is important to account for size because of different breeds and different body weights of the studied dogs (Boström et al., 2014; Henderson et al., 2015). Therefore, to further test our hypothesis we measured the CSA of the vertebral body caudal endplate (Cain et al., 2016; Henderson et al., 2015). The results from the Muscle:VB ratio were mostly in line with the results from the Muscle:Disc ratio, with a discrepancy only for the Mm. multifidi in both analyses and for the combined Mm. multifidi, *M. longissimus* dorsi, and M. iliocostalis measurements in the analysis of only affected discs. One possible explanation for this is that the CSA of Mm. multifidi is so small in relation to the CSA of the disc and vertebral endplate that this measurement is affected more by small variations.

We also hypothesized that the muscular fat infiltration would be increased in dogs with compressive IVDD relative to dogs with non-compressive ANNPE or FCE. The Muscle:fat ratio was higher in the compressive group than in the non-compressive group in the analysis of all measured intervertebral discs and in that of only affected intervertebral discs. This means higher signal intensity on MRI in the muscles of compressive IVDD dogs, indicating increased fat infiltration. The estimation of muscular fat infiltration using the signal intensity on the MRI T1 sequence is recognized in both humans (Crawford et al., 2017; Elliott et al., 2006; D'Hooge et al., 2012) and dogs (Boström et al., 2014; Lerer et al., 2015). Previous research has interpreted increased signals in epaxial muscle T1-weighted sequences as fat infiltration in Dachshunds with IVDD (Boström et al., 2014) and in non-chondrodystrophic dogs with general spinal pathology (Lerer et al., 2015). Denervation may explain our findings. Acute denervation (<1 month) causes oedema-like changes in muscle on MRI, normal T1-weighted signals, and increased T2-weighted signals (Kamath et al., 2008). Subacute to chronic denervation (1–6 months) appears as increased signals on both T1- and T2-weighted MRI because of an increase in extracellular water and progressive fat infiltration (Kamath et al., 2008). A recent study found hyperintense signals on T2-weighted sequences and hypo to isointense signals on T1-weighted sequences in the epaxial muscles of dogs with acute thoracolumbar disc extrusion (Trampus et al., 2018). Such MRI signal alterations correspond to oedema in the muscles, and denervation oedema as a consequence of the spinal cord injury is the explanation suggested (Trampus et al., 2018). The reasoning above (Kamath et al., 2008; Trampus et al., 2018) and the prolonged duration of clinical signs in our study dogs with compressive IVDD support the theory that the changes detected on MRI are fat infiltration.

Eleven of the dogs in the compressive group had multiple lesions (2–4 lesions), and they suffered from prolonged neurological deficits and almost all of them were paraparetic. Most of the dogs in the non-compressive group presented with only one lesion site and acute onset of non-ambulatory paraparesis or -plegia. This difference between the groups may explain the visible atrophy of the epaxial muscles in the compressive dogs. Previous research has found that dogs with

**Table 3**
Correlations between clinical parameters and muscular variables.

| Muscle ratio | Age | | Duration of clinical signs (days) | | Neurological grade | |
|---|---|---|---|---|---|---|
| | r | p | r | p | r | p |
| EPAX:DISC (all) | −0.387 | 0.034* | −0.532 | 0.003* | 0.306 | 0.100 |
| EPAX:DISC (lesion) | −0.481 | 0.007* | −0.439 | 0.015* | 0.219 | 0.245 |
| MM:DISC (all) | −0.401 | 0.028* | −0.384 | 0.036* | 0.194 | 0.304 |
| MM:DISC (lesion) | −0.491 | 0.006* | −0.269 | 0.113 | 0.104 | 0.584 |
| MMEPAX:DISC (all) | −0.332 | 0.073 | −0.487 | 0.006* | 0.289 | 0.121 |
| MMEPAX:DISC (lesion) | −0.404 | 0.027* | −0.425 | 0.019* | 0.213 | 0.258 |
| EPAX:VB (all) | −0.323 | 0.081 | −0.538 | 0.002* | 0.260 | 0.166 |
| EPAX:VB (lesion) | −0.335 | 0.071 | −0.473 | 0.008* | 0.214 | 0.255 |
| MM:VB (all) | −0.336 | 0.070 | −0.376 | 0.041* | 0.081 | 0.669 |
| MM:VB (lesion) | −0.361 | 0.050 | −0.360 | 0.050 | 0.046 | 0.810 |
| EPAXM:VB (all) | −0.373 | <0.0001* | −0.486 | <0.0001* | 0.186 | 0.012* |
| EPAXM:VB (lesion) | −0.340 | 0.066 | −0.410 | 0.024* | 0.213 | 0.259 |
| EPAX:FAT (all) | 0.335 | 0.071 | 0.393 | 0.031* | −0.072 | 0.706 |
| EPAX:FAT (lesion) | 0.419 | 0.021* | 0.390 | 0.033* | −0.001 | 0.997 |
| MM:FAT (all) | 0.387 | 0.035* | 0.424 | 0.020* | −0.039 | 0.837 |
| MM:FAT (lesion) | 0.442 | 0.015* | 0.496 | 0.005* | −0.028 | 0.884 |
| MMEPAX:FAT (all) | 0.402 | 0.028* | 0.416 | 0.022* | −0.029 | 0.881 |
| MMEPAX:FAT (lesion) | 0.455 | 0.011* | 0.376 | 0.041* | 0.005 | 0.980 |

Correlation between age, duration of clinical signs (days), and neurological grade for muscle variables ('all' indicates all measured vertebral discs, 'lesion' indicates only affected vertebral discs). *P*-values are based on Spearman correlation coefficient (r) and statistical significance ($p < 0.050$) is indicated with an asterisk.

degenerative annular protrusion have longer duration of neurological deficits and lower grade of neurological dysfunction than dogs with nuclear extrusions (Macias et al., 2002). The dogs in the compressive group had a longer duration of clinical signs (63.6 ± 85.5 days vs. 1.2 ± 0.5 days) than the dogs in the non-compressive group, and correlation analysis revealed that as the duration of clinical signs increases the fat infiltration increases for all muscles assessed.

The age of the dogs may have influenced the fat infiltration. The dogs with compressive IVDD were significantly older than the non-compressive ANNPE/FCE dogs, and a positive correlation emerged between age and muscular fat infiltration, indicating more fat infiltration in the muscles of older dogs. Furthermore, Dachshunds with compressive IVDD with a mean age of 7.3 ± 2.3 years had greater fat infiltration in their muscles than dogs in the younger control group (Boström et al., 2014). In humans, fat infiltration increases with age, especially in the back muscles relative to the muscles of the leg (Dahlqvist et al., 2017). Age has also been associated with a significant increase of fat infiltration in the lumbar multifidus over time (Fortin et al., 2013). Further, age is reported to explain some of the variance in volume and fat infiltration of the Mm. multifidi lumborum and M. erector spinae in humans (Valentin et al., 2015). Future veterinary studies should consider the possible influence of age on muscle measurements.

So, why is it important to investigate potential atrophy in the epaxial muscles of dogs with spinal pathologies? Current treatment recommendations for large dogs with compressive IVDD are crate rest and restricted activity for several weeks (Fenn et al., 2016). Our results support previous reports suggesting that epaxial muscles in dogs with spinal disease are compromised in both size and composition (Boström et al., 2014; Cain et al., 2016; Henderson et al., 2015; Lerer et al., 2015; Trampus et al., 2018). In humans, it is well known that substantial muscle atrophy occurs after only five days of disuse (Wall et al., 2014). For dogs with compressive IVDD and chronic progressive clinical signs, the denervation atrophy resulting in secondary disuse atrophy may be evident in the epaxial muscles already before they receive diagnosis and treatment. Recovery with rest and controlled walking only may result in further atrophy, potentially even limiting improvement of muscle function and strength. A large number of dogs with annular protrusions receiving medical treatment deteriorate within one year of diagnosis and some are euthanized (Crawford and De Decker, 2017; Fenn et al., 2016,). Also, not all cases are surgical candidates due to surgery being challenging to perform because of multiple lesions, spinal cord changes, or financial constraints (Fenn et al., 2016; Macias et al., 2002), hence, there is a need for complementary therapy such as physiotherapy.

Current physiotherapy strategies for large dogs suffering from compressive IVDD focus on maintaining and restoring hind limb function (Hodgson et al., 2017; Olby et al., 2005; Sims et al., 2015). Retraining the epaxial muscles receives little attention. In humans with back pain, muscle function is improved with individually designed training, specifically targeting the spinal musculature, aiming to improve neuromuscular control, endurance, and strength during functional tasks required in everyday life (Liddle et al., 2007; Inani and Selkar, 2013; Richards et al., 2013). In dogs, the epaxial muscles work as dynamic stabilizers of the spine, controlling spinal flexion and rotation (Webster et al., 2014). Electromyographic activity in the Mm. multifidi and *M. longissimus* lumborum is higher during trotting than in walking or standing (Schilling and Carrier, 2010). Therefore, the physiotherapy approach for humans could be implemented in dogs as follows: ensuring normal and pain-free range of motion in the entire vertebral column with flexion and extension exercises with combined rotation of the spine mimicking functional tasks (reaching for food or a toy) to engage all of the epaxial muscles in the thoracolumbar area. Slow, controlled trotting on the lead introduced early (at 4–6 weeks postoperatively) would stimulate epaxial muscle activity. Early consultation with a physiotherapist may encourage controlled and safe general exercise during the early postoperative period, possibly preventing further atrophy. However, it must be noted that the reason for resting dogs with compressive IVDD is to prevent the risk of more disc material extruding into the spinal canal, resulting in further deterioration in neurological function. It is important that future research aims to determine whether controlled physiotherapy impacts this risk.

Our study has some limitations. Firstly, the retrospective nature of the study rendered comparison with healthy dogs impossible. Considering the non-compressive character and the acute non-progressive onset of clinical signs in FCE and ANNPE, these patients provide a suitable control group for dogs suffering from compressive IVDD. However, a definitive diagnosis of ANNPE and FCE was not made in these dogs because final diagnosis is difficult to achieve without a post-mortem or confirmation during surgery (Fenn et al., 2016). Further, signal intensity changes in the epaxial muscles have been reported recently also in dogs with FCE (Martens et al., 2018), and further studies using healthy control dogs are needed to explain the noted signal intensity changes. Another consideration when comparing and interpreting MR images from IVDD and ANNPE/FCE dogs is that in clinical practice the ANNPE/FCE dogs are scanned immediately, while the dogs with compressive IVDD may have had their clinical signs for some time before being scanned. Another matter that cannot be overlooked is that two dogs in the ANNPE/FCE group had incidental findings of secondary chronic disc protrusions in addition to the primary acute ANNPE/FCE for which they were treated. This may have influenced the results.

Secondly, there was no information available on dogs' exercise level or parameters that could allow for body mass index calculations, thus, it was not possible to account for these variables' effects on the muscles. Thirdly, the averaged measurements provide only general information about the muscles in the thoracolumbar region. Analysing segments separately and caudally to the lesion would provide more specific information on denervation atrophy, as the nerve root innervates the muscle bulk caudal to its foramen (Evans, 1993). Unfortunately, such specific analyses were not feasible in our retrospective study with multiple lesion sites for several dogs, making it difficult to reliably determine the clinically significant lesion.

Strengths of this study are the reliable measurement method used (Boström et al., 2014) and the fact that several slices from the same segments were measured. Measuring the CSA at only one site provides limited information on the muscle at that particular segment. Also considering the naturally occurring anatomical variation between spinal segments (Webster et al., 2014), measuring the CSA at several segments and then calculating the average, as was done here, reflects reality more appropriately. Future studies could consider muscle volume measurements since volume provides a more truthful estimate of the muscles' contractility (Valentin et al., 2015). Unfortunately, this was not possible here due to the retrospective nature of this study allowing the evaluated images to have variability in slice thickness and spacing between slices. Further, the orientation of the transverse slice in relation to the spine may affect the CSA measurement and this issue should be addressed in future prospective studies on this topic.

There is a need for developing specific progressive targeted training and controlled general exercise regimes for large breed dogs recovering from compressive IVDD that would not provoke symptoms but would be safe and efficient. Interventions to preserve and restore spinal musculature may prevent recurrence of pain episodes, enhance physical activity, and improve quality of life. Future prospective study designs using validated quantification and healthy control dogs would be beneficial to investigate the effects of such strategies.

## 5. Conclusions

We conclude that there is increased fat infiltration in the Mm. multifidi and *M. longissimus* dorsi muscles in dogs with compressive IVDD relative to dogs with non-compressive ANNPE or FCE. This is likely a consequence of denervation atrophy and secondary general disuse, caused by prolonged, mild neurological deficits and possible pain. These results further establish the Muscle:fat ratio as a useful measure of

muscular fat infiltration, while disc CSA may not be a reliable measurement in standardization for size in dogs with IVDD.

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## Declaration of Competing Interest

The authors have no competing interests to declare.

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