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# Comparison of cross sectional area and fat infiltration of the epaxial muscles in dogs with and without spinal cord compression



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## ABSTRACT

This study investigated the cross sectional area (CSA) and fat infiltration of the epaxial muscles in Dachshunds with compressive spinal cord lesions due to intervertebral disc herniation (IVDH) and in dogs with non-compressive spinal cord lesions with fibrocartilaginous embolism (FCE). The CSA and fat infiltration of the multifidi and longissimus dorsi muscles were determined from T1 weighted magnetic resonance images. Difference in CSA and fat infiltration between the lesion- and non-lesion side in the Dachshunds was assessed using mixed model analysis. Difference in CSA and fat infiltration between Dachshunds and FCE dogs was analysed with independent sample t-tests.

There was no difference in CSA or fat infiltration between sides in the Dachshunds. FCE dogs had greater CSA (multifidus  $P = 0.036$ , longissimus  $P < 0.001$ ) and less fat infiltration compared to Dachshunds (longissimus  $P = 0.017$ ). Duration of neurological deficits, age, body size and conformation are likely to have influenced the difference between the groups.

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## 1. Introduction

Intervertebral disc herniation (IVDH) is a common problem in companion dogs (Bergknut et al., 2012; Packer et al., 2013) with a clear breed predisposition for degeneration of the intervertebral discs (Hansen type I disc degeneration) among Dachshunds (Jensen et al., 2008). In this disease, the nucleus pulposus degenerates and mineralises. The annulus fibrosus may rupture and extrusion of the nucleus pulposus into the vertebral canal causes compression of the nerve roots or spinal cord (Hoerlein, 1952). In Dachshunds this occurs most frequently in the thoracolumbar area, accompanying signs may be acute onset of pain, paresis or plegia (Hoerlein, 1952). Research in veterinary medicine has focused on the pathogenesis (Spitzbarth et al., 2011), histopathology (Henke et al., 2013), surgical treatment (Laitinen and Puerto, 2005), recovery to ambulation (Olby et al., 2003) and prognosis for recovery (Davis and Brown, 2002; Ruddle et al., 2006).

Human low back pain (LBP) is defined as local or radiating, often non-specific pain arising from the lower part of the spine and is a frequent reason for persistent disability and early retirement (Luomajoki, 2010). Human research has used magnetic resonance

imaging (MRI) (Kader et al., 2000; Kang et al., 2007) to investigate the cross sectional area (CSA) and fat infiltration of the back muscles in subjects with LBP in order to design effective management strategies for this problem (Hides et al., 1994; Mannion et al., 2009). Decrease of multifidus and erector spinae muscle CSA and fat infiltration of the muscle tissue are indicators of atrophy and associated with LBP (Kang et al., 2007; Kjaer et al., 2007). Humans with both acute (Hides et al., 1994) and chronic (Hides et al., 2008a) LBP have displayed decreased CSA in the multifidus muscle ipsilateral to the focus of painful symptoms. Asymptomatic subjects have significantly larger multifidi muscles compared to those with LBP (Hides et al., 2008a). Reduced CSA and dysfunction of the multifidus can predispose to recurrence of the symptoms (Hides et al., 2001); however, the changes may persist even though the pain has resolved (Hides et al., 1996). The multifidi are considered one of the main stabilisers of the human spine (Moseley et al., 2002, 2003) and specific training of the multifidi muscles has reduced the recurrence of LBP (Hides et al., 2001), restored the CSA of the multifidi muscles and relieved the symptoms (Danneels et al., 2000; Hides et al., 2008b).

The anatomy (Evans 1993a), innervation (Bogduk, 2005a; Kottlors and Glocker, 2008) and function (Ritter et al., 2001; Schilling and Carrier, 2009) of the canine multifidi and longissimus dorsi muscles are similar to that in humans. Multifidi muscle atrophy occurs also in horses with back pain (Stubbs et al., 2010) and one scientifically tested management strategy of back pain both in humans (Hides et al., 2008b; Mannion et al., 2009) and horses (Stubbs et al., 2011)

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**Table 1**  
Descriptive statistics of the studied dogs.

Group	Breed (n)	Gender (n)	Lesion category (n)	Age*	Weight
IVDH	Dachshunds (52)	Male (25) Female (27)	Acute compressive (32) Chronic compressive (20)	7.3 ± 2.3	7.2 ± 2.5
FCE	Various breeds (12) <sup>a</sup>	Male (7) Female (5)	Acute non-compressive (12)	5.6 ± 2.4	10.1 ± 3.9

The breed, gender, lesion category, mean and standard deviation of the age and weight in the studied dogs. <sup>a</sup>Wales terrier (1), Border terrier (1), Bishon frisé (1), Dachshund (1), Staffordshire bullterrier (1), Whippet (1), Cocker spaniel (1), Chihuahua (1), Lhasa apso (1), German spitz (1), Mixed breed (2).

\*Significant difference between IVDH and FCE groups (P = 0.021).

is specific retraining of the multifidi muscles. To date, there are no reports on atrophy, CSA or fat infiltration of epaxial muscles in dogs with or without signs of spinal cord disorders that would provide justification for therapeutic exercises.

The aim of this study was to determine the relationship between compressive and non-compressive spinal cord lesions in dogs weighing ≤15 kg on the thoracolumbar epaxial muscles. The multifidi and longissimus dorsi muscles at the level of the T10–L3 vertebrae were assessed for CSA and fat infiltration in Dachshunds with compressive spinal cord lesions, IVDH and in dogs with non-compressive spinal cord lesions diagnosed with FCE.

## 2. Materials and methods

### 2.1. Subjects

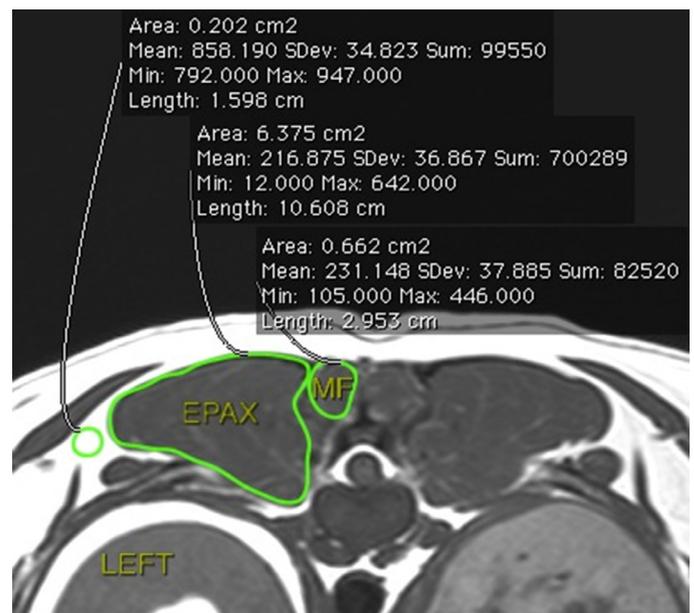
The ethics and welfare committee of the authors' institution approved this study. As there were no canine studies the sample size was approximated using information from a human study that evaluated the CSA in several spinal segments (Hides et al., 2008a, Table 1), suggesting 8–24 dogs in each group (based on 5% type I error and 80% power). Fifty-two client owned Dachshunds and 12 control dogs undergoing spinal MRI at the Royal Veterinary College (RVC) between 2003 and 2010 were retrospectively reviewed from patient records for inclusion in the study. Inclusion criteria were to be a Dachshund with a myelopathy secondary to IVDH, localised between the third thoracic and third lumbar (T3–L3) spinal cord segment and diagnosed by MRI. Dogs with other causes for T3–L3 myelopathy or evidence for previous spinal cord compression with or without surgery were excluded from the study. Control dogs were dogs of ≤15 kg with a T3–L3 myelopathy diagnosed on MRI to have a FCE and no evidence for compression of the spinal cord. All dogs had to have good quality transverse T1-weighted MRI images of the spine between the 10th thoracic and the 3rd lumbar vertebrae (T10–L3). Lesion side, lesion site, age, breed, sex, body weight, neurological grading at the time of presentation, duration of neurological deficits and duration of pain reported by the owner prior to presentation was retrieved from the patient records.

If the side of lesion was not found in the patient records, the ECVN Diplomat in the author team (ED) confirmed the side of lesion from the MRIs. Many IVDH lesions were partly ventral and it was decided to classify the lesion as 'right sided' if the lesion was right and ventral and 'left sided' if left and ventral. The lesion was classified as 'midline', only if the lesion was purely midline. The neurological grade at the time of presentation was retrieved from patient records and where not written, it was determined retrospectively based on the Modified Frankel Score used previously by Van Wie et al., 2013.

Based on the nature of the spinal cord lesion the dogs were categorised into three groups: Dachshunds with acute compressive lesions (duration of acute neurological signs less than 7 days prior to presentation), Dachshunds with chronic compressive lesions (>7 days) and other small breed dogs with acute non-compressive lesions (FCE dogs).

### 2.2. Methodology

All MR images were obtained with a 1.5 Tesla scanner (Phillips Intera, Phillips Medical, Reigate, UK). Segments from the T10–L3 vertebrae were analysed using a dedicated DICOM viewer (Osirix, Pixmeo, Bernex, Switzerland) from T1 sequences (TE 8–120, TR 400.00–3680.79, slice thickness 2.5–4.0 mm and gap 2.8–4.4 mm). The muscle measurements were made in random order at the level of the disc at each segment as previously reported (Kang et al., 2007). The CSA of the disc (DISC CSA) was measured in the same image as the muscular measurements (Fig. 1). The CSA of the multifidi muscles (MMCSA), the CSA of the longissimus dorsi and the iliocostal muscles (EPAXCSA) and the CSA of multifidi, longissimus and iliocostal muscles combined (MMEPAXCSA) were measured bilaterally by drawing a region of interest (ROI) around the muscle, tracing the muscle margins visible on the MRI (Fig. 1). The multifidus muscle was measured alone, whereas the longissimus dorsi muscle and the iliocostal muscle were measured together forming the epaxial muscle measurement, as it was difficult to distinguish between these muscles. To compensate for possible discrepancy in body weight and body conformation between IVDH Dachshunds and FCE dogs, a muscle to disc ratio (Kang et al., 2007) was calculated for all muscle variables (Multifidus CSA:Disc (MM:DISC), Epaxial CSA:Disc



**Fig. 1.** The measurements. The CSA measurements on the left side at T12–13 in a FCE dog. The Muscle:Fat ratio was calculated using the hyperintensity mean of muscle and the hyperintensity mean of fat. (i.e. Muscle:Fat ratio for the left side Epaxial measurement: Epaxial hyperintensity mean (216.875)/Fat hyperintensity mean (858.190) = EPAX:FAT).

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