



Magnetic Resonance Imaging Comparison of Intervertebral Disc Degeneration of Normal and Disc Diseases in Dogs

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Abstract This study compared the degree of disc degeneration of intervertebral disc between normal disc and disc disease using Magnetic Resonance images of tholacolumbar and lumbar vertebrae in dogs. The sample population consisted of 72 dogs and 188 intervertebral discs. These dogs were divided into four groups according to MRI criteria: normal, disc protrusion, disc extrusion, and fibrocartilaginous embolism. The Pfirrmann classification developed by Pfirrmann for use in human medicine was used to assess the degree of disc degeneration. Statistical analysis revealed that disc diseases had a significant difference in the degree of disc degeneration compared to normal discs in the intervertebral disc. Fibrocartilaginous embolism was found to have a relatively low disc degenerative change compared to two other disc disease groups, disc protrusion and disc extrusion. Disc degeneration in the disc extrusion group was slightly higher than that in the disc protrusion group, although the difference between the two groups was not statistically significant.

Key words dogs, intervertebral disc, magnetic resonance imaging, degeneration.

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Introduction

Intervertebral disc (IVD) is a connective structure between vertebral bodies that plays an important role in the maintenance of a deformable space which facilitates flexibility of the vertebral column. IVD also acts as a shock absorber in the assimilation of compressive forces (20). IVD is a disc-shaped structure composed of nucleus pulposus (NP), annulus fibrosus (AF), and cartilage end plates (CEP) (6,20,24). The NP is a soft, highly hydrophilic material contained within the central zone of the IVD. The AF surrounds the NP. The CEP is at each end of the vertebral centrum (20).

Degeneration of the IVD is inevitable in the aging process. Progressive pathological changes begin to appear in structure (1,21). The onset of disc degeneration in humans starts with the loss of diffusional capacity of blood vessels of the vertebral endplate that can act as a nutrient supplier to the nucleus pulposus. This degeneration of the blood supply may result from many mechanisms in humans. However, most disc degeneration in dogs is deeply associated with multigenetic elements (21,22).

Magnetic resonance imaging (MRI) is the most suitable imaging modality for dogs in assessing IVDs as it provides relatively complete anatomical description of the spinal cord and vertebral column (3,23). Intervertebral disc disease (IVDD) is the most common canine spinal cord disease. IVDD is a spinal cord compression disease due to herniation of IVDs (4,19,29). In disc extrusion, also known as Hansen type-I, the spinal cord is pressed with rapid extrusion of disc materials into the spinal canal through a ruptured annulus fibrosus. Disc protrusion, also known as Hansen type-II, presses on the spinal cord with a focal and gradual protrusion of the disc into the spinal canal (8,13,19,29). Fibrocartilaginous embolism (FCE) is a disease in which fibrocartilaginous materials that are histologically and histochemically similar to the NP of the IVD cause vascular occlusion in the spine, resulting in ischemic necrosis of dependent regions in the spinal cord parenchyma (12,14).

Previous studies evaluating IVD degeneration have not compared normal discs with disc diseases. Thus, the aim of this retrospective study was to compare degrees of degeneration in the IVD of those with normal discs and those with disc diseases including protrusion, extrusion, and FCE using thoracolumbar and lumbar MRI images.

Materials and Methods

This study was performed prospectively for dogs that underwent thoracolumbar and lumbar MRI at Veterinary Medical Teaching Hospitals of Gyeongsang National University

from March 2016 to June 2019.

Dogs were divided into four groups by MRI criteria: normal, disc protrusion, disc extrusion, and FCE. MRI diagnostic criteria for disc diseases were as follows. Normal was defined if there was no specific finding in the IVD. Selection for disc protrusion was based on the presence of focal midline or dorsolateral extension of the disc margin and focal rupture of the annulus fibrosus (Fig. 1A, B) (17). Disc extrusion was diagnosed if disc materials herniated through all layers of the annulus fibrosus (Fig. 1C, D) (17). FCE was selected a focal T2 hyperintense intramedullary lesion and T1 hypointense or isointense and mono-, hemi-, tetraparesis or tetraplegia upon physical examination (Fig. 1E, F) (18,24). The FCE group evaluated the disc closest to the lesion. Dogs with both disc protrusion and extrusion were excluded (Fig. 2).

MRI (APERTO 0.4 tesla, Hitachi Medical Co., Tokyo, Japan and Vantage Elan 1.5 tesla, Toshiba medical Co., Tokyo, Japan) was performed in dorsal recumbency for dogs to evaluate thoracolumbar and lumbar regions. MRI acquired T2-weighted (T2W) spin echo sequences. The sagittal plane was acquired with 2-3 mm of thickness. All MRI images were reviewed with a DICOM view (RadiAnt DICOM Viewer, version 4.6.9; Medixant Inc., Poznan, Poland) and evaluated by a single observer (J.S).

Pfarrmann classification developed by Pfarrmann in 2001 (26) for use in human medicine was used to evaluate the degree of disc degeneration from MRI images. We used the Pfarrmann classification which had five grades ranging from grade 1 to grade 5 using an algorithm based on disc structure, distinction of nucleus and annulus, signal intensity, and height of IVD (Table 1) (7,26,32).

Statistical analysis was performed using the SPSS statistical computer program (SPSS for Windows, Release 25.0, standard version, SPSS Inc., Chicago, Illinois, USA). IVD degeneration differences of the four groups were statistically analyzed using Kruskal-Wallis test followed by post-hoc Mann Whitney U-test. In the statistical test, a p-value less than 0.008 was considered to indicate a significant difference.

Results

Seventy-two dogs (48 males and 24 females) of 17 breeds were enrolled in this study. Represented breeds were Maltese (n = 24), Shih-tzu (n = 9), Dachshund (n = 7), Mixed breeds (n = 7), Poodle (n = 5), Pekingese (n = 4), Cocker Spaniel (n = 2), Miniature Pinscher (n = 2), Chihuahua (n = 2), Pomeranian (n = 2), Boston Terrier (n = 2), Beagle (n = 1), Schnauzer (n = 1), Yorkshire Terrier (n = 1), Spitz (n = 1), Coton de tular (n = 1), and Pungsan dog (n = 1). They aged 1 year to 14 years (mean

age \pm standard deviation: 6.8 ± 3.22 years). Their body weights were 2 kg to 15.7 kg (mean weight \pm standard deviation 5.7 ± 2.88 kg).

The sample population consisted of 188 IVDs from 72 dogs. Selected dogs and IVDs were divided into four groups: normal (10 dogs, 79 discs), disc protrusion (30 dogs, 72 discs), disc extrusion (24 dogs, 26 discs), and FCE (8 dogs, 11 discs). The number of disc degenerative grades in each group was as follows:

normal group, grade 1 ($n = 76$; 96%) and grade 2 ($n = 3$; 4%); disc protrusion group, grade 2 ($n = 4$; 6%), grade 3 ($n = 27$; 37%), and grade 4 ($n = 41$; 57%); disc extrusion group, grade 3 ($n = 5$; 19%) and grade 4 ($n = 21$; 81%); and FCE group, grade 1 ($n = 2$; 18%), grade 2 ($n = 2$; 18%), grade 3 ($n = 5$; 46%), and grade 4 ($n = 2$; 18%) (Table 2).

Disc protrusion was the most frequently identified ($n = 15$) in L1-2 and L2-3. Disc extrusion was the most frequently identified ($n = 10$) in the L1-2. FCE was similar in all regions, although it was most frequently identified ($n = 4$) in the T12-13 (Fig. 3).

Comparison of disc degeneration among the four groups identified statistically significant differences in all groups except for disc degeneration between the disc protrusion group and the disc extrusion group. There were significant differences in disc degeneration between normal group (1.04 ± 0.19) and each disc disease group, between protrusion (3.51 ± 0.61) and FCE (2.64 ± 1.03), and between extrusion (3.8 ± 0.40) and FCE ($p < 0.008$) (Fig. 4).

Discussion

IVDD can occur in all dog breeds, although it is more common in chondrodystrophic (CD) dogs (29). CD dogs have disproportionately short limbs. They are mainly characterized by long bone endochondral ossification disorders. Representative CD dogs include Dachshund, Bulldog, Shih-tzu, Miniature Pinscher, Pekingese, Beagle, and Cocker Spaniel (19,29). Most breeds used in this study except Maltese were CD dogs. The most common types of dogs in Korea have been reported to be Maltese, Shih-Tzu, Yorkshire Terrier, and Poodle in order (27). Based on this, it was postulated that the ratio of Maltese in this study was the highest.

Previous studies on IVDD in the thoracolumbar of dogs have reported the most degenerative changes in T12-13 and L3-4 among 417 IVDs (7). In this study, both protrusion and extrusion showed most degenerative changes in the L1-2

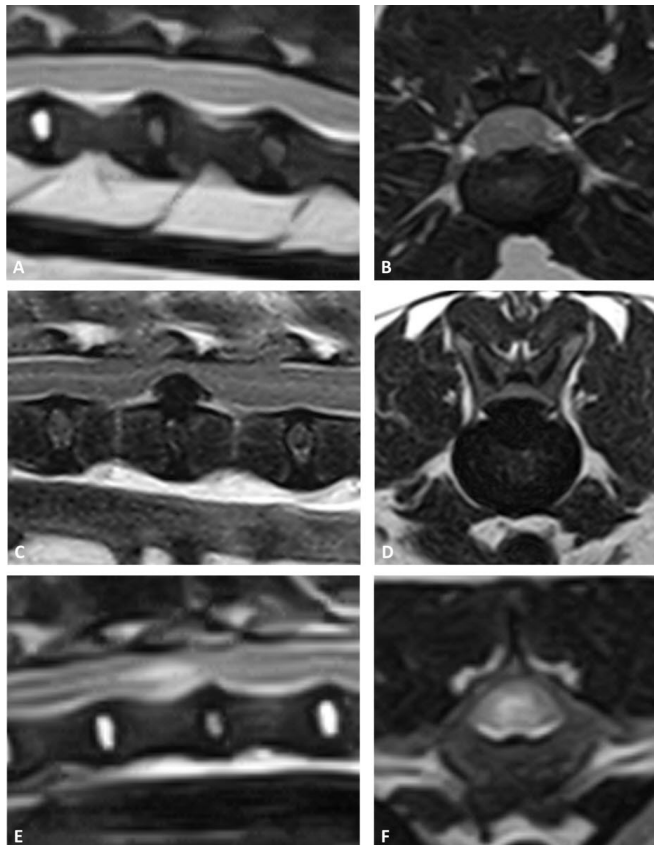


Fig. 1. T2W sagittal and transverse images of lumbar intervertebral disc protrusion (A, B), extrusion (C, D), and fibrocartilaginous embolism (E, F).

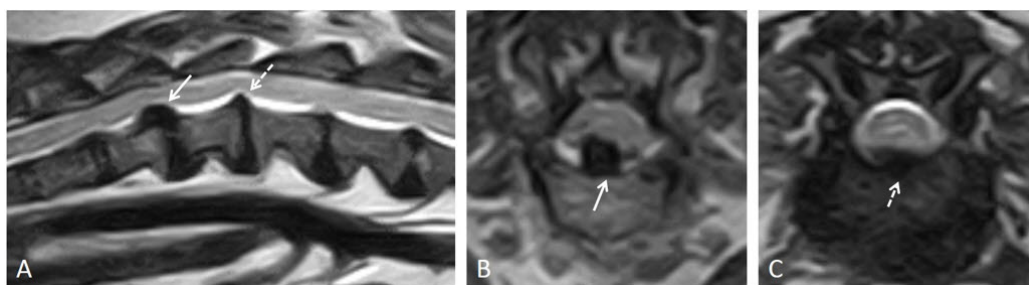


Fig. 2. Exclusion criteria due to two intervertebral disc diseases on magnetic resonance imaging. (A) Sagittal T2-weighted image (T2WI). (B, C) Transverse T2-weighted images (T2WI). Disc extrusion (arrows) and disc protrusion (dashed arrows) are shown.

Table 1. Pfirrmann’s classification of disc degeneration

Grade	Structure	Distinction of nucleus and anulus	Signal intensity	Height of intervertebral disc
I	Homogeneous, bright white	Clear	Hyperintense, isointense to cerebrospinal fluid	Normal
II	Inhomogeneous with or without horizontal bands	Clear	Hyperintense, isointense to cerebrospinal fluid	Normal
III	Inhomogeneous, gray	Unclear	Intermediate	Normal to slightly decreased
IV	Inhomogeneous, gray to black	Lost	Intermediate to hypointense	Normal to moderately decreased
V	Inhomogeneous, black	Lost	Hypointense	Collapsed disc space

Table 2. Number of degenerative grades of intervertebral discs in each group

Grade	Normal (10 dogs, 79 discs)	Protrusion (30 dogs, 72 discs)	Extrusion (24 dogs, 26 discs)	FCE (8 dogs, 11 discs)
I	76 (96%)	0	0	2 (18%)
II	3 (4%)	4 (6%)	0	2 (18%)
III	0	27 (37%)	5 (19%)	5 (46%)
IV	0	41 (57%)	21 (81%)	2 (18%)
V	0	0	0	0

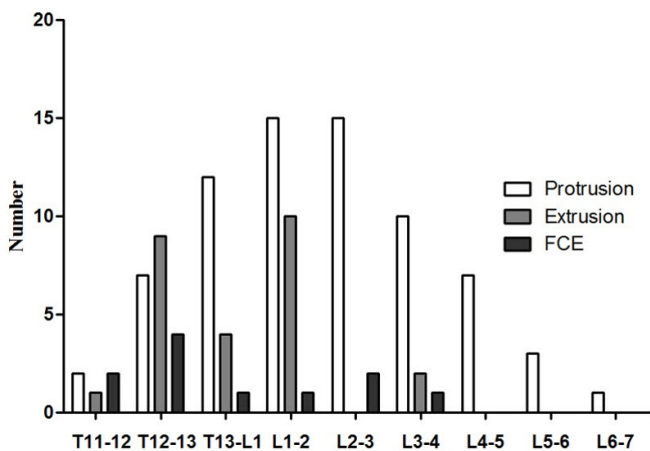


Fig. 3. The number by region of intervertebral disc in different disc diseases, such as protrusion, extrusion, and FCE.

region, with FCE found sporadically. This might be due to the limited number of discs analyzed.

IVDs can stabilize the vertebral column by holding adjacent vertebrae to each other. At the same time, they allow flexibility by enabling vertebral movement to absorb and distribute weight that is loaded on the spine (9,28). With increasing age, degenerative changes occur in IVDs as blood supply becomes insufficient (9,21,31). The histological structure of IVDs in humans is similar to that in dogs for different stages of IVD degeneration. The nucleus pulposus and anulus fibrosus in humans and dogs have similar form, density and cell pop-

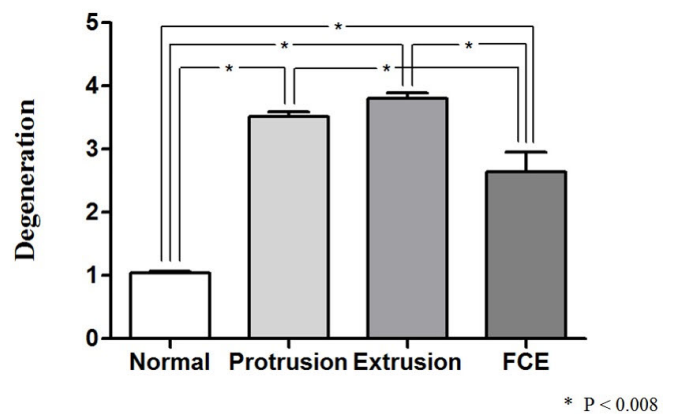


Fig. 4. Comparison of disc degeneration among normal, protrusion, extrusion, and fibrocartilaginous embolism (FCE) groups. Asterisk (*) indicates statistically different at $p < 0.008$.

ulations (5). In humans, with increasing age, the boundary between nucleus and anulus fibrosus becomes less distinct and fibrosis takes place as proteoglycan and water contents in the nucleus pulposus decrease (31). With loss of proteoglycan, the osmotic pressure in the disc decreases, resulting in an inability to maintain hydration under pressure. Compared to a disc of a normal age, degenerate discs have lower water contents and a tendency to quickly lose height and fluid and to bulge under pressure (2,31). Also, the anulus fibrosus becomes irregular and weaker while the collagen and elastine become disorganized (2,9,31). The height of a disc does not

show a major decrease with age, while discs that have aged due to degenerative changes might have collapsed annuli (2). In humans, it is known that some degenerative changes occur before disc herniation. Disc herniation resulting from migration of degenerated fragments of nucleus pulposus through tears in the annulus fibrosus has been reported (25,31). The present study also showed a significant difference in disc degenerative change between the normal group and disc-associated disease groups.

Recent studies have shown that IVD protrusion and extrusion have different clinical characteristics, although they are pathologically similar (6,17). In this study, no significant difference in disc degenerative change was noted between the disc protrusion group and the disc extrusion group. Thus, there was no difference in disc degeneration between the two types of disc herniation. However, the ratio of grade 4 extrusion was slightly higher than that of protrusion, although their difference was not statistically significant. Disc extrusion is characterized by sudden inflow of calcified and fragmented nucleus pulposus into the vertebral canal. Protrusion has been reported to be caused by gradual hypertrophy and hyperplasia of the annulus fibrosus (17,19,29). This is considered to be slightly higher in the degree of degeneration of the extrusion than protrusion.

FCE is a disease in which fibrocartilaginous materials cause vascular occlusion in the spine, resulting in ischemic damage of the spinal cord parenchyma. Typical clinical findings are hyperacute onset (<6 h) of non-painful, non-progressive, and asymmetric or symmetric motor dysfunction (11,18). The definitive diagnosis of FCE is obtained upon histopathological diagnosis of the spinal cord after autopsy (16,30). The pathway by which fibrocartilaginous materials are taken into the vascular system remains unclear, although several hypotheses exist, including direct penetration of materials of degenerated disc into spinal vessels, penetration into newly formed inflamed vessels in a degenerated IVD, penetration into remaining embryonic vessels in the nucleus, and penetration into sinusoidal vessels in the bone marrow (10,18). In previous studies, 24% of dogs identified as FCE at necropsy have been reported to have IVD lesions (10). In horses with FCE confirmed, small fibrocartilaginous fragments attached on the dura mater over the infarcted part have also been observed microscopically (15). Based on this, it could be postulated that there is a direct relation between embolism and disc degeneration, although the frequency and degree of degeneration would be low. In this study, there was a significant difference in the degree of disc degeneration between FCE and disc diseases such as protrusion and extrusion. Based on this, it could be surmised that FCE has a relatively low degree of degeneration compared to

disc protrusion and extrusion.

This study has several limitations. First, the number of patients in the FCE group was relatively low compared to those in other groups. Therefore, we could not accurately evaluate the degree of disc degeneration in FCE. Further studies with more dogs are needed. Second, histopathological examination was not performed for the spinal cord of the FCE group. Finally, selected dogs of the protrusion and extrusion groups were not confirmed surgically. However, the diagnosis was based on the criteria of protrusion and extrusion using MRI in previous study (17).

This study demonstrates that disc diseases in the IVD have a significant difference in the degree of disc degeneration compared to normal discs on MRI. FCE was confirmed to have a relatively low degree of disc degenerative change than the other two groups of disc diseases, disc protrusion and disc extrusion.

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Conflicts of Interest

The authors have no conflicting interests.

References

1. Adams MA, Dolan P. Intervertebral disc degeneration: evidence for two distinct phenotypes. *J Anat* 2012; 221: 497-506.
2. Adams MA, Roughley PJ. What is intervertebral disc degeneration, and what causes it? *Spine (Phila Pa 1976)* 2006; 31: 2151-2161.
3. Adams WH, Daniel GB, Pardo AD, Selcer RR. Magnetic resonance imaging of the caudal lumbar and lumbosacral spine in 13 dogs (1990–1993). *Vet Radiol Ultrasound* 1995; 36: 3-13.
4. Bergknut N, Egenvall A, Hagman R, Gustås P, Hazewinkel HA, Meij BP, et al. Incidence of intervertebral disk degeneration-related diseases and associated mortality rates in dogs. *J Am Vet Med Assoc* 2012; 240: 1300-1309.
5. Bergknut N, Rutges JP, Kranenburg HJ, Smolders LA, Hagman R, Smidt HJ, et al. The dog as an animal model for intervertebral disc degeneration? *Spine (Phila Pa 1976)* 2012; 37: 351-358.
6. Bergknut N, Smolders LA, Grinwis GC, Hagman R, Lagerstedt AS, Hazewinkel HA, et al. Intervertebral disc degeneration in the dog. Part 1: anatomy and physiology of the intervertebral disc and characteristics of intervertebral disc degeneration. *Vet J* 2013; 195: 282-291.
7. Besalti O, Pekcan Z, Sirin YS, Erbas G. Magnetic resonance imag-

- ing findings in dogs with thoracolumbar intervertebral disk disease: 69 cases (1997-2005). *J Am Vet Med Assoc* 2006; 228: 902-908.
8. Braund KG, Ghosh P, Taylor TK, Larsen LH. Morphological studies of the canine intervertebral disc. The assignment of the beagle to the achondroplastic classification. *Res Vet Sci* 1975; 19: 167-172.
 9. Buckwalter JA. Aging and degeneration of the human intervertebral disc. *Spine (Phila Pa 1976)* 1995; 20: 1307-1314.
 10. Cauzinille L. Fibrocartilaginous embolism in dogs. *Vet Clin North Am Small Anim Pract* 2000; 30: 155-167, vii.
 11. Cauzinille L, Kornegay JN. Fibrocartilaginous embolism of the spinal cord in dogs: review of 36 histologically confirmed cases and retrospective study of 26 suspected cases. *J Vet Intern Med* 1996; 10: 241-245.
 12. De Risio L, Platt SR. Fibrocartilaginous embolic myelopathy in small animals. *Vet Clin North Am Small Anim Pract* 2010; 40: 859-869.
 13. Dewey CW. Surgery of the thoracolumbar spine. In: Fossum TW, editor. *Small animal surgery*. 4th ed. St. Louis: Elsevier. 2013: 1508-1528.
 14. Dyce J, Houlton JEF. Fibrocartilaginous embolism in the dog. *J Small Anim Pract* 1993; 34: 332-336.
 15. Fuentealba IC, Weeks BR, Martin MT, Joyce JR, Wease GS. Spinal cord ischemic necrosis due to fibrocartilaginous embolism in a horse. *J Vet Diagn Invest* 1991; 3: 176-179.
 16. Gandini G, Cizinauskas S, Lang J, Fatzner R, Jaggy A. Fibrocartilaginous embolism in 75 dogs: clinical findings and factors influencing the recovery rate. *J Small Anim Pract* 2003; 44: 76-80.
 17. Gomes SA, Volk HA, Packer RM, Kenny PJ, Beltran E, De Decker S. Clinical and magnetic resonance imaging characteristics of thoracolumbar intervertebral disk extrusions and protrusions in large breed dogs. *Vet Radiol Ultrasound* 2016; 57: 417-426.
 18. Gorgas DS. Ischemic myelopathy, spinal cord hemorrhage, myelomalacia. In: Mai W, editor. *Diagnostic MRI in dogs and cats*. Boca Raton: CRC Press. 2018: 565-569.
 19. Hansen HJ. A pathologic-anatomical study on disc degeneration in dog, with special reference to the so-called enchondrosis intervertebralis. *Acta Orthop Scand Suppl* 1952; 11: 1-117.
 20. Humzah MD, Soames RW. Human intervertebral disc: structure and function. *Anat Rec* 1988; 220: 337-356.
 21. Jeffery ND, Levine JM, Olby NJ, Stein VM. Intervertebral disk degeneration in dogs: consequences, diagnosis, treatment, and future directions. *J Vet Intern Med* 2013; 27: 1318-1333.
 22. Kepler CK, Ponnappan RK, Tannoury CA, Risbud MV, Anderson DG. The molecular basis of intervertebral disc degeneration. *Spine J* 2013; 13: 318-330.
 23. Macias C, Mckee WM, May C, Innes JF. Thoracolumbar disc disease in large dogs: a study of 99 cases. *J Small Anim Pract* 2002; 43: 439-446.
 24. Mai W. Magnetic resonance imaging and computed tomography features of canine and feline spinal cord disease. In: Thrall D, editor. *Textbook of veterinary diagnostic radiology*. 7th ed. St. Louis: Elsevier. 2018: 271-304.
 25. Moore RJ, Vernon-Roberts B, Fraser RD, Osti OL, Schembri M. The origin and fate of herniated lumbar intervertebral disc tissue. *Spine (Phila Pa 1976)* 1996; 21: 2149-2155.
 26. Pfirrmann CW, Metzendorf A, Zanetti M, Hodler J, Boos N. Magnetic resonance classification of lumbar intervertebral disc degeneration. *Spine (Phila Pa 1976)* 2001; 26: 1873-1878.
 27. Podberscek AL. Good to pet and eat: the keeping and consuming of dogs and cats in South Korea. *J Soc Issues* 2009; 65: 615-632.
 28. Sakai D, Andersson GB. Stem cell therapy for intervertebral disc regeneration: obstacles and solutions. *Nat Rev Rheumatol* 2015; 11: 243-256.
 29. Smolders LA, Bergknut N, Grinwis GC, Hagman R, Lagerstedt AS, Hazewinkel HA, et al. Intervertebral disc degeneration in the dog. Part 2: chondrodystrophic and non-chondrodystrophic breeds. *Vet J* 2013; 195: 292-299.
 30. Ueno H, Shimizu J, Uzuka Y, Kobayashi Y, Hirokawa H, Ueno E, et al. Fibrocartilaginous embolism in a chondrodystrophoid breed dog. *Aust Vet J* 2005; 83: 142-144.
 31. Urban JP, Roberts S. Degeneration of the intervertebral disc. *Arthritis Res Ther* 2003; 5: 120-130.
 32. Yu LP, Qian WW, Yin GY, Ren YX, Hu ZY. MRI assessment of lumbar intervertebral disc degeneration with lumbar degenerative disease using the Pfirrmann grading systems. *PLoS One* 2012; 7: e48074.