Intervertebral disc decompression following endplate damage: implications for disc degeneration depend on spinal level and age

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Abstract

Study Design Mechanical and morphological studies on cadaveric spines.

Summary of Background Data Disc degeneration can be initiated by damage to a vertebral body endplate, but it is unclear why endplate lesions, and patterns of disc degeneration, vary so much with spinal level and age.

Objective Explain how spinal level and age influence disc degeneration arising from endplate fracture.

Methods 174 cadaveric 'motion segments', from T7-8 to L5-S1 and aged 19-96 yrs, were subjected to controlled compressive overload to damage a vertebral body. 'Stress profilometry' was performed before and after damage in order to quantify changes in intradiscal pressure (IDP), and compressive stresses in the annulus. 86 of the undamaged vertebral bodies were then sectioned in the mid-sagittal plane, and the thickness of the central bony endplate was measured from microradiographs. Regression analysis was used to compare the relative influences of spinal level, age, disc degeneration and gender on results obtained.

Results Compressive overload caused endplate fracture at an average force of 3.4 kN, and reduced vertebral body height by an average 1.88 mm. Pressure loss in the adjacent nucleus pulposus decreased from 93% at T8-9 to 38% at L4-5 (R^2 = 22%, P<0.001), and increased with age (R^2 = 19%, P<0.001) especially in male specimens. Stress concentrations in the posterior annulus increased following endplate fracture, with the effect being greatest at upper spinal levels (R^2 = 7%, P<0.001). Endplate thickness increased by approximately 50% between T11 and L5 (R^2 = 21%, P<0.001).

Conclusion Endplate fracture creates abnormal stress distributions in the adjacent intervertebral disc, increasing the risk of internal disruption and degeneration. Effects are greatly reduced in the lower lumbar spine, and in young specimens, primarily because of differences in nucleus volume, and materials properties, respectively. Disc degeneration between L4 and S1 may often be unrelated to endplate fracture.

Key Points

1. Disc degeneration can be initiated by damage to a vertebral endplate, but why should disc and endplate lesions vary so greatly with spinal level and age?
2. Experiments on 174 cadaveric motion segments (aged 19-96 yrs, from T7-8 to L5-S1) showed that compressive overload *always* damaged a vertebral body endplate and decompressed the adjacent intervertebral disc.
3. The severity of disc decompression depended primarily on spinal level, decreasing linearly from 93% at T8-9 to 38% at L4-5 ($R^2 = 22\%$, $P<0.001$), and was lower in young male specimens. Variations appeared to depend on systematic differences in nucleus volume and endplate thickness.
4. Results suggest that disc degeneration at L4-5 and L5-S1 may often be unrelated to endplate fracture.

*Mini Abstract (50 words)

Précis

Experiments on cadaveric spines showed that endplate fracture always decompresses the adjacent nucleus pulposus, and concentrates compressive stress in the posterior annulus fibrosus. Effects are much reduced in the lower lumbar spine, and in young specimens, as a result of increased nucleus volume and thicker endplates. Disc degeneration between L4 and S1 may often be unrelated to endplate fracture.

Introduction

Recent population studies show a strong dose-response relationship between intervertebral disc degeneration and chronic low back pain.[1-3] However, the relationship is weak if degeneration is defined in terms of age-related water loss,[4-5] and strong if 'degeneration' involves structural features such as radial fissures,[6-7] posterior herniation,[8] endplate defects,[9] and reinnervation[10-11].

This disagreement highlights a growing problem: 'disc degeneration' encompasses a range of features, each with its own risk factors, and opportunities for intervention. Only when the disc finally collapses can the process be likened to a single disease, and by then it may be too late for effective intervention apart from surgery. Clearly there is a need to distinguish between separate disc degeneration 'phenotypes' earlier in the degeneration process, so that distinct pathological processes can be treated or prevented more effectively.

With this in mind, we recently contrasted 'endplate-driven' and 'annulus-driven' disc degeneration.[12] The former is characterised by an endplate fracture, inwards collapse of the annulus, high heritability, and a distribution predominantly in the upper lumbar and thoracic spine. In contrast, 'annulus-driven' degeneration is characterised by a radial fissure and/or disc prolapse, low heritability, and a distribution predominantly in the lower lumbar spine (L4-S1). The distinction is simplistic (endplates *can be* damaged at L4 and L5, and upper lumbar discs *can* herniate) but it is consistent with a diverse range of evidence, and it suggests why spinal level should exert such a strong influence on spinal pathology.[13-15] One experimental finding in particular supports this 'two-phenotypes' concept: severe mechanical loading is more likely to create radial fissures and herniation in lower lumbar discs than in upper lumbar discs.[16-17] However, there is no corresponding evidence that upper lumbar and

thoracic discs are more vulnerable to endplate fracture, and its sequellae, than are lower lumbar discs.

We hypothesise that endplate damage arising from excessive spinal compression causes major decompression of thoracic and upper lumbar discs, but only minor decompression of lower lumbar discs. Nucleus decompression following endplate fracture has been shown to cause progressive internal disruption in cadaveric discs,[18] leading to realistic disc degeneration in an animal model.[19]

Materials and Methods

Cadaveric material Thoracolumbar spines from donated cadavers were stored at -20^0 C before being dissected into 'motion segments' comprising one or two discs and their adjacent vertebrae.[20-23] Details of the 174 tested discs are summarised in **Table 1**.

Stress profilometry Each motion segment was mounted in plaster (**Figure 1**) and loaded on a materials testing machine.[20] During testing, specimens were wrapped in plastic film to minimise water loss. A moderate compressive force (typically 1 kN) was applied for 1-2 hours in order to dehydrate the disc by an amount similar to that seen in-vivo during a day's activity,[24] hence ensuring that disc hydration was well within the normal physiological range. After the creep loading period, a pressure transducer (side-mounted in a 1.3 mm-diameter needle) was inserted into the disc and pulled across its mid-sagittal diameter to measure the distribution of compressive "stress".[25] These "stress profiles" were usually obtained at a compressive load of 1kN with the specimen positioned either in the neutral position (0^o) or in 2^o of extension, to simulate typical erect postures in life. Vertical and horizontal stress was measured in successive tests by rotating the transducer needle about its axis. Intradiscal pressure (IDP) in the nucleus, and peak compressive stresses in the annulus (over and above nucleus pressure), were then measured from the 'profiles'.[22] For some

specimens, the compressive force during creep loading and stress profilometry was changed to 0.5 kN or 2 kN, depending on specimen size and age, but all intradiscal measurements were subsequently scaled to an applied load of 1 kN for comparison. Stress profilometry has been validated[26-28] and linearity between measured 'stress' and applied load has been demonstrated.[29]

Compressive overload With the specimen positioned in moderate flexion (to simulate the flat back of a weight-lifter[30]) the compressive force was increased by moving the ram of the testing machine upwards at 3mm/s. Loading was removed at a pre-determined maximum displacement which depended on specimen size and age, and the force-deformation graph was inspected for evidence that the yield point had been exceeded.[20] If not, the specimen was loaded to a higher maximum displacement. Eventually, the first sign of injury was revealed by a reduction in gradient of the force-deformation graph. Endplate damage was confirmed by radiographs,[31] and quantified by repeating the loading cycle up to the failure load, and noting the extent to which it was shifted along the X (displacement) axis.[20] The X-shift (in mm), recorded at a load of 1 kN, indicated motion segment height loss.

Optical measurements of vertebral body deformation To help interpret the Dartec-based measurements of motion segment height loss, a single-camera MacReflex system was used to measure deformation of the damaged vertebral body independently of deformation of the disc, the non-damaged vertebral body, and the apparatus[23]. This technique can be subject to large errors if vertebral damage disturbs the reflective markers, which is more likely to occur in smaller thoracic vertebrae. Therefore, these measurements were obtained in 15 specimens from T11-12 to L4-5. In these specimens, three pairs of reflective markers were attached to the lateral cortex of each vertebral body tested (Figure 1) so that anterior, middle and posterior vertebral body heights could be measured,[23] to an accuracy of 10μm.[32] Height

measurements were compared before and after compressive damage in order to calculate cortical height loss in mm.[23]

Specimen morphology After testing, each specimen was dissected and photographed to confirm the site(s) of fracture. The macroscopic appearance of each disc, and its height on pre-fracture radiographs, were used to grade disc degeneration from 1 to 4, using the first four points on a scale described previously.[33]

Endplate thickness 86 of the non-damaged vertebral bodies, sampled so that all age groups and spinal levels were represented, were sectioned in the sagittal plane. Subsequent microradiographs of 2mm-thick slices were analysed to measure the thickness of the cranial and caudal bony endplates at 10 equidistant sites along the mid-sagittal section. Because fracture primarily affects the central endplate, thickness was averaged across sites 4, 5, 6 and 7, which lie adjacent to the inner annulus and nucleus.[34]

Statistical analyses Linear regression was used to determine which variable factors had the greatest influence on disc decompression and vertebral damage (height loss).

Results

Initial measurements on 174 undamaged specimens showed that IDP averaged 0.88 MPa (for a reference compressive force of 1kN) and decreased from 0.98 MPa in 'grade 1' discs to 0.47 MPa in severely degenerated 'grade 4' discs (**Table 2**, column 4**)**. IDP also decreased with spinal level, from 1.56 MPa at T7-8, to 0.73 MPa at L5-S1.

Compressive overload always damaged a bony endplate, although some specimens showed additional vertebral damage to the anterior cortex and/or trabeculae. The compressive force at the initiation of damage averaged 3.4 kN (range 0.9 - 11.6 kN). Motion segment height loss averaged 1.88 mm and was greatest in old female specimens and in those with degenerated discs (Table 2, column 5). Height loss averaged 2.27 mm at T7-8, but was less

in the lumbar spine, falling to 0.46 mm at L5-S1. Optical measurements showed that height loss averaged 17%, 11% and 4% in the anterior, middle and posterior regions of the lateral vertebral body cortex of the damaged vertebra, indicating slight anterior wedging.

Vertebral damage reduced IDP in the adjacent disc, by an average 0.54 MPa, which is equivalent to a 62% loss. In contrast, maximum compressive stress in the posterior annulus (over and above nucleus pressure) increased by 0.74 MPa. Nucleus decompression varied greatly with spinal level, from 1.28 MPa at T7-8 to 0.24 MPa at L4-5 (Table 2, column 7). The influence of spinal level did not depend much on whether the discs were degenerated (grades 3 and 4) or not (grades 1 and 2), as shown in **Figure 2**, or on whether decompression was measured in absolute (MPa) or relative (%) terms (Table 2). Nucleus decompression also increased linearly with age in male specimens, whereas nearly all female specimens over the age of 40 yrs were substantially decompressed (**Figure 3**). Similar age-related trends were present if decompression was quantified in absolute or relative terms.

Univariate regression analysis (**Table 3)** showed that the greatest influence on the extent of vertebral damage was spinal level, which explained 20% of variance in all specimens, and 34% in specimens with non-degenerated discs (Table 3, row 3). Vertebral damage also increased with age (Table 3, row 1) and with female gender (Table 2, column 5). Damage was in turn a major influence in disc decompression, explaining 20% of the fall in IDP in all specimens, and 47% in specimens with non-degenerated discs (Table 3, row 14). Age explained 19% of the variance in % disc decompression. However, absolute changes in intradiscal stresses (MPa) following vertebral damage depended less on age than on initial IDP (Table 3, rows 13 & 18) which itself decreased with age and disc degeneration (Table 2, column 4). Peak compressive stress in the posterior annulus (relative to IDP) increased in proportion to the fall in IDP (Table 3, row 20), and increased most when initial IDP was high

(Table 3, row 18). Changes in peak compressive stresses in the *anterior* annulus (not shown) were variable, and unrelated to other variables in Table 3.

Multiple linear regression revealed the greatest influences on disc decompression. If the three independent variables (age, gender and spinal level) were forced into the analysis, they explained 38% of the variance in 'Fall in IDP (MPa)'. However, adding 'Initial IDP (MPa)', 'Damage (mm)', and 'disc degeneration (graded 1-4)' to the model enabled it to explain 71% of the variance in disc decompression (**Table 4)**. Additional multivariate analyses revealed that 'Initial IDP (MPa)' itself depended on spinal level, age, and gender (in decreasing order of importance) but the influence of disc degeneration was marginal.

Thickness of the bony endplate is compared at various spinal levels (**Figure 4)**. Values in Figure 4 represent the average thickness (of both endplates of each vertebral body) in the central region, adjacent to the nucleus and inner annulus, because this is where fracture generally occurs[34]. Thickness increased by approximately 50% between T11 and L5 (R^2 = 21%, P<0.001). Endplate thickness was measured on 86 *undamaged* vertebrae, and so was not compared directly with failure characteristics such as specimen strength.

Discussion

Summary of findings Compressive overload always damaged a vertebral body endplate and decompressed the adjacent nucleus pulposus. Decompression was greatest in old specimens, in those with degenerated discs, and in the upper lumbar and thoracic spine. Thickness of the bony endplates increased between T11 and L5.

Strengths and weaknesses of the investigation. Many specimens were tested so that influences such as age and spinal level might be distinguished. All techniques have been validated, as discussed above. Vertebral endplate fractures were obtained by compressing them via the adjacent disc, and their radiographic appearance (which included some anterior

wedging) was similar to fractures seen clinically.[31] The use of cadaveric tissues introduces little artefact,[35] and measurements of intradiscal pressure (IDP) are similar to those made in-vivo,[36-38] showing similar variation with degeneration.[39]

Working with dead tissues ensures that long-term consequences of endplate damage can only be estimated. Also, specimens were not distributed evenly between spinal levels and age groups, with relatively few specimens from L5-S1, and few young specimens from thoracic levels (Table 1). Measurements of vertebral damage would have been influenced by the loading protocol, but their dependence on age and gender (Table 3) accurately reflects the fact that, in life, vertebral collapse is greatest in old female vertebrae.

Relationship to previous studies. Compressive overload has been shown primarily to damage the vertebral endplate[40-42] and its supporting trabeculae.[43] Spinal flexion and endplate fracture both transfer load-bearing to the anterior vertebral cortex,[23, 44] explaining why anterior cortical damage was substantial in the present experiments. Endplate damage decompresses the adjacent disc,[45-46] leading to internal collapse of the inner annulus,[18] and increased radial bulging of the outer annulus,[43] both of which are common features of disc degeneration in-vivo.[47-48] The novelty of the present experiment is to show how disc decompression varies with factors such as spinal level and age.

Explanation of findings Damaged endplates bulge more into the vertebral body[43] allowing more space for the disc nucleus. This reduces nucleus pressure, which is sensitive to small % changes in volume.[49] Greater damage causes greater nucleus decompression, and correspondingly greater peak stresses in the annulus, as load-bearing is shifted from nucleus to annulus, and to the neural arch.[22] The following discussion suggests why this mechanism depends primarily on spinal level, age/degeneration and gender, and secondarily on factors such as 'Damage (mm)' which themselves depend on the primary variables.

The influence of spinal level has several likely causes, the most important of which is probably the greater *height* of lower lumbar discs. Disc height increases substantially in the lower thoracic spine[50] and by 100-150% between T7-8 and L5-S1,[51] so nucleus volume will increase by at least this amount. Decompression following endplate damage is proportional to nucleus volume, and so will be reduced at lower lumbar levels. The greater height of lower lumbar discs leads to greater vertical deformations and radial bulging under load,[51] enhancing the disc's ability to equalise compressive stress and reducing nucleus decompression. Increasing disc cross-sectional area at lower spinal levels[40] may also exert an influence, because it will cause the 1 kN load to give rise to a lower 'initial IDP', which in turn may cause a smaller pressure drop following endplate damage. However, endplate area increases by only 17% between L1 and L5[52], whereas disc decompression across these same levels decreased by 59% (calculated from Table 2 column 7) so the influence of endplate area is not paramount. Finally, thicker vertebral endplates in the lower lumbar spine (**Figure 4**) may minimise endplate deformations and disc pressure changes following endplate damage.

The influence of age (Figure 3) is also complex. Nucleus pressure decreases with age and disc degeneration[39],[25] and a lower 'Initial IDP' will naturally cause a greater % fall in IDP following endplate damage for the same absolute pressure drop. Age also makes vertebrae more brittle, so that they lose more height when damaged and cause greater disc decompression. Hence, the marked gender differences shown in Figure 3 could be explained in terms of increasing bone fragility in women after the menopause. Annulus tissue also stiffens with age as a result of non-enzymatic glycation,[53] so an old stiff annulus may be better able to stress-shield the nucleus than the soft hydrated annulus of a young non-degenerated disc, which can deform and equalise stress.

The influence of age and spinal level on nucleus decompression can be compared as follows: age probably exerts its influence by increasing annulus stiffness and bone fragility, whereas

spinal level probably exerts its influence via annulus height and nucleus volume. Although both influences are large, it is evident from Table 2 that only spinal level has a major influence on *absolute* pressure loss in the nucleus (in MPa) and on absolute stress increases in the posterior annulus. Absolute changes in intradiscal stress may be more important than % changes when it comes to driving disc disruption and degeneration.

The influence of gender can be explained in terms of increased bone fragility in women, and also on the reduced size and endplate area in female spines, which would increase initial disc pressure and lead to greater damage and subsequent pressure changes in the disc (Table 2).

Clinical implications. Disc decompression and 'internal disc disruption' is common in the thoracolumbar spine.[47] From a biological point of view, low pressure in the nucleus and increased stresses in the annulus will inhibit disc cell metabolism[54] and increase synthesis of matrix-degrading enzymes.[55-56] In this way, endplate failure would initiate biological degenerative changes in the disc. This mechanism of disc degeneration has been demonstrated in animals,[19, 57] explained by organ culture[56] and mathematical models,[58] and confirmed in humans.[59]

Decompression, and loss of disc height, following endplate fracture are associated with an increase in neutral zone and hence with instability.[20] Greater decompression of thoracic and upper lumbar discs therefore explains why degenerative disc narrowing and osteophytes, which are typical in this region of the spine,[50, 60] are so closely associated with large centrally-located Schmorl's nodes.[14] Some nodes may be congenital,[41] but others are related to loading[15] and represent calcification around a vertical herniation of nucleus pulposus. Thoracic disc degeneration tends not to lead to severe radial bulging,[50] possibly because the discs are inherently narrow and better stress-shielded by the neural arch, and thoracic disc degeneration is only 'modestly' associated with back pain.[61]

The situation is very different between L4 and S1, where Schmorl's nodes are relatively uncommon[15, 62] and less associated with disc degeneration.[14] This can be explained by the present results: thicker endplates between L4 and S1 reduce the risk and size of vertical disc herniations, and the hence the tendency for the disc to degenerate via this mechanism. Most lower lumbar endplate lesions are characterised as 'erosions/calcification' rather than 'Schmorl's nodes/fractures',[15] and are associated with inflammation in the vertebral body. [63-64] They may reflect posterior disc herniations in which displaced annulus strips cartilage from the bony endplate,[65-66] allowing inflammatory reactions to occur. Many disc herniations involve disruption of cartilage and bone on the postero-lateral margins of the vertebral body.[67]

These interpretations support the concept of two disc degeneration phenotypes[12]: 'endplate-driven' degeneration in the upper lumbar and thoracic spine which is initiated by endplate damage as simulated in the present experiment; and 'annulus driven' degeneration at L4-5 and L5-S1 which is initiated by nucleus tissue herniating through the annulus. Both of the initiating lesions decompress the nucleus, making it less likely that the other type of lesion could occur in the same disc.

Unanswered questions and future research. Longitudinal clinical studies are required to prove that different mechanisms lead to disc degeneration at L4-S1 compared to higher spinal levels.

Conclusions Even minor damage to a vertebral body endplate can decompresses the adjacent disc, allowing the annulus to collapse inwards. The effect is small in young and lower lumbar discs, possibly because they have a larger nucleus volume, and thicker endplates. In life, degeneration of L4-S1 discs may often be unrelated to endplate fracture.

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Table 1: Details of 174 cadaveric specimens tested. Values refer to the number of specimens in each sub-group.

Number of specimens tested (total =174)									
Spinal Level	n	Grade of disc degeneration				Age (yrs)		Gender	
		1	2	3	4	<=60	>60	Male	Female
T7-8	6		4	2		2	4	2	4
T8-9	4			4		1	3	2	2
T9-10	9		5	3	1	3	6	3	6
T10-11	12		5	7		3	9	6	6
T11-12	17		8	5	4	1	16	10	7
T12-L1	21		11	9	1	4	17	12	9
L1-2	27		11	13	3	4	23	12	15
L2-3	33	2	15	14	2	13	20	20	13
L3-4	23		7	10	6	5	18	13	10
L4-5	20	4	8	7	1	12	8	13	7
L5-S1	2		1	1		2		2	
Sub-totals	174	6	75	75	18	50	124	95	79

Table 2: Summary of results for various specimen groups. Values indicate the mean. n = number of specimens in each group. DD = grade of disc degeneration. The fall in intradiscal pressure (IDP) following endplate damage is given in absolute units (MPa), and also as a % of the Initial IDP before damage. "PA stress rise" = absolute increase in peak compressive stress (relative to IDP) measured in the posterior annulus, caused by endplate damage.

1	2	3	4	5	6	7	8
Specimen groups	n	Age (yrs)	Initial IDP (MPa)	Damage (mm)	% Fall in IDP	Fall in IDP (MPa)	PA stress rise (MPa)
All	174	71	0.88	1.88	62	0.54	0.74
Male	95	70	0.79	1.73	57	0.42	0.64
Female	79	72	0.99	2.06	68	0.69	0.85
Age<=60	50	48	1.09	1.48	46	0.54	0.73
Age > 60	124	80	0.79	2.05	69	0.54	0.74
DD - 1	6	24	0.98	0.81	5	0.05	0.03
DD - 2	75	65	1.00	1.79	56	0.57	0.79
DD - 3	75	77	0.86	1.98	68	0.60	0.71
DD - 4	18	84	0.47	2.21	80	0.34	0.86
T7-8	6	72	1.56	2.27	87	1.28	1.82
T8-9	4	74	1.27	2.16	93	1.19	1.09
T9-10	9	68	1.23	2.71	85	1.01	1.56
T10-11	12	74	0.87	2.44	89	0.79	0.49
T11-12	17	80	0.89	2.38	77	0.66	0.95
T12-L1	21	74	1.00	1.94	63	0.60	0.79
L1-2	27	77	0.85	1.89	71	0.59	0.85
L2-3	33	68	0.83	1.58	49	0.37	0.60
L3-4	23	71	0.64	1.92	48	0.29	0.43
L4-5	20	56	0.74	1.09	38	0.24	0.35
L5-S1	2	47	0.73	0.46	5	0.02	0.25

Table 3 Summary of univariate linear regression results. A negative sign indicates an inverse relationship. Gender was coded 0 (female) and 1 (male), and spinal levels from T7-8 to L5-S1 were coded from 1 to 11. Dependent (Y) variables represent changes following endplate damage.

Row no.	Dependent (Y) variable	Independent (X) variable	All discs (n=174)		Non-degenerated discs (n=81)	
			Rsq	P	Rsq	P
1	Damage (mm)	age	17	<0.001	14	0.001
2		gender	-4	0.011	-15	<0.001
3		spinal level	- 20	<0.001	-34	<0.001
4		initial IDP	0	NS	8	0.009
5	% Fall in IDP	age	19	<0.001	19	<0.001
6		gender	-3	0.031	-9	0.006
7		spinal level	-22	<0.001	-32	<0.001
8		initial IDP	0	NS	1	NS
9		damage	37	<0.001	51	<0.001
10	Fall in IDP (MPa)	age	2	NS	1	NS
11		gender	-9	<0.001	-12	0.002
12		spinal level	-33	<0.001	-35	<0.001
13		initial IDP	49	<0.001	44	<0.001
14		damage	20	<0.001	47	<0.001
15	PA stress rise (MPa)	age	0	NS	0	NS
16		gender	-1	NS	-4	NS
17		spinal level	-7	<0.001	-7	0.021
18		initial IDP	25	<0.001	28	<0.001
19		damage	4	0.007	10	0.003
20		fall in IDP	27	<0.001	31	<0.001

Table 4 The best predictive model for disc decompression, obtained using multiple linear regression, explained 71% of the variance in 'Fall in IDP (MPa)'. Spinal level was coded from 1 (T7-8) to 11 (L5-S1), gender was coded 0 (female) and 1 (male), and disc degeneration graded from 1-4. Standardised (Std) coefficients indicate the relative importance of each influence. A '–ve' sign indicates an inverse relationship.

All discs (n=174) $\mathbf{R^2 = 71\%}$		
	Std coefficients	P
Initial IDP (MPa)	0.662	<0.001
Damage (mm)	0.244	<0.001
Spinal level	(-) 0.179	<0.001
Age (yrs)	0.138	0.016
Gender	(-) 0.066	NS
Disc Degeneration	0.058	NS

Figure Captions

Figure 1 Cadaveric thoracolumbar motion segments were secured in cups of dental plaster, and compressed by means of two rollers, which maintained a neutral posture without inhibiting any settling movements in the horizontal plane. Black circles represent reflective markers that were attached to the lateral vertebral body cortex in some specimens: these enabled deformations to be measured optically, independent of deformations of other tissues and apparatus. The distribution of compressive stress was measured within the intervertebral disc by pulling a miniature pressure transducer along its mid-sagittal diameter.

Figure 2 Disc decompression (Fall in IDP) following endplate damage decreased regularly from T7-8 down to L5-S1, regardless of whether the disc was degenerated or not. 81 of the 174 specimens were non-degenerated (disc grade 1 or 2). Error bars indicate the standard error of the mean (SEM).

Figure 3 Disc decompression following vertebral endplate damage increased with age, but only in male specimens. In contrast, most female specimens aged over 40 yrs were substantially decompressed. Error bars indicate the SEM.

Figure 4 Average thickness of the vertebral body endplate was minimal at T11, and increased linearly from T11 to L5 ($R^2 = 21\%$, $P<0.001$). Data refer to 80 undamaged vertebrae.

Figures (TIFF, EPS, or PPT only)

Figure 1

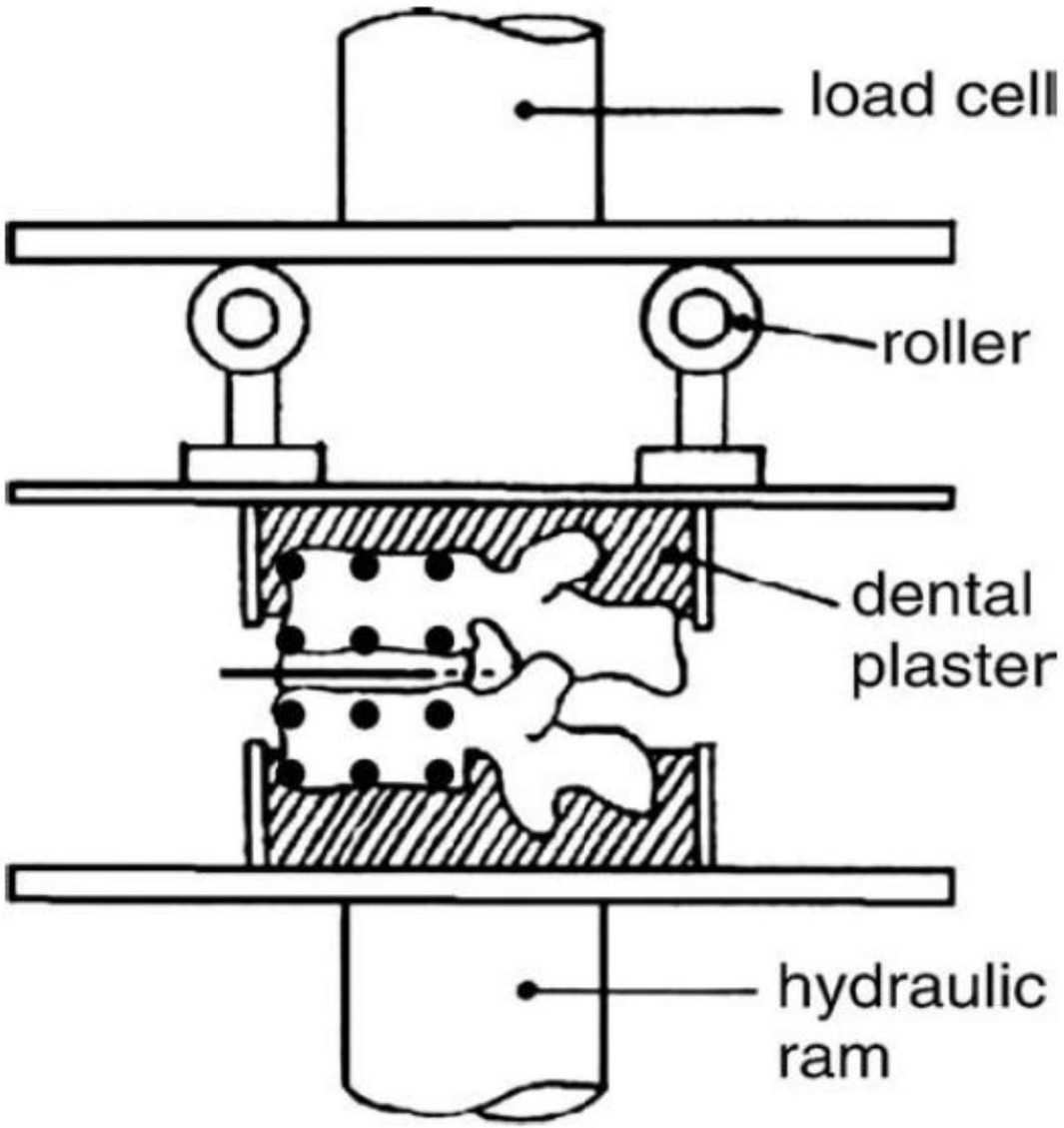


Figure 2

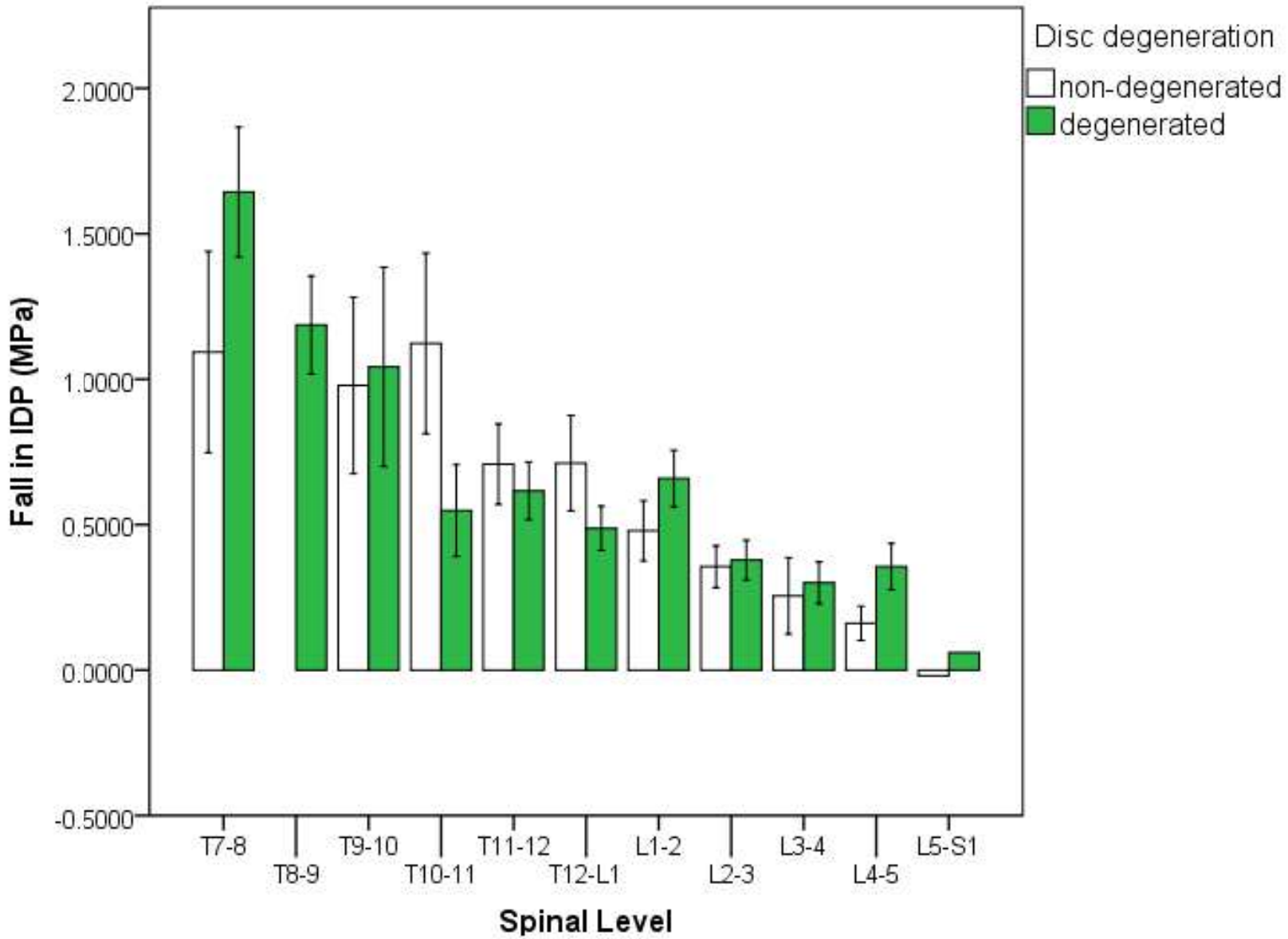

Disc degeneration
non-degenerated
degenerated
Fall in IDP (MPa)
2.0000
1.5000
1.0000
0.5000
0.0000
-0.5000
T7-8
T8-9
T9-10
T10-11
T11-12
T12-L1
L1-2
L2-3
L3-4
L4-5
L5-S1
Spinal Level

Figure 3

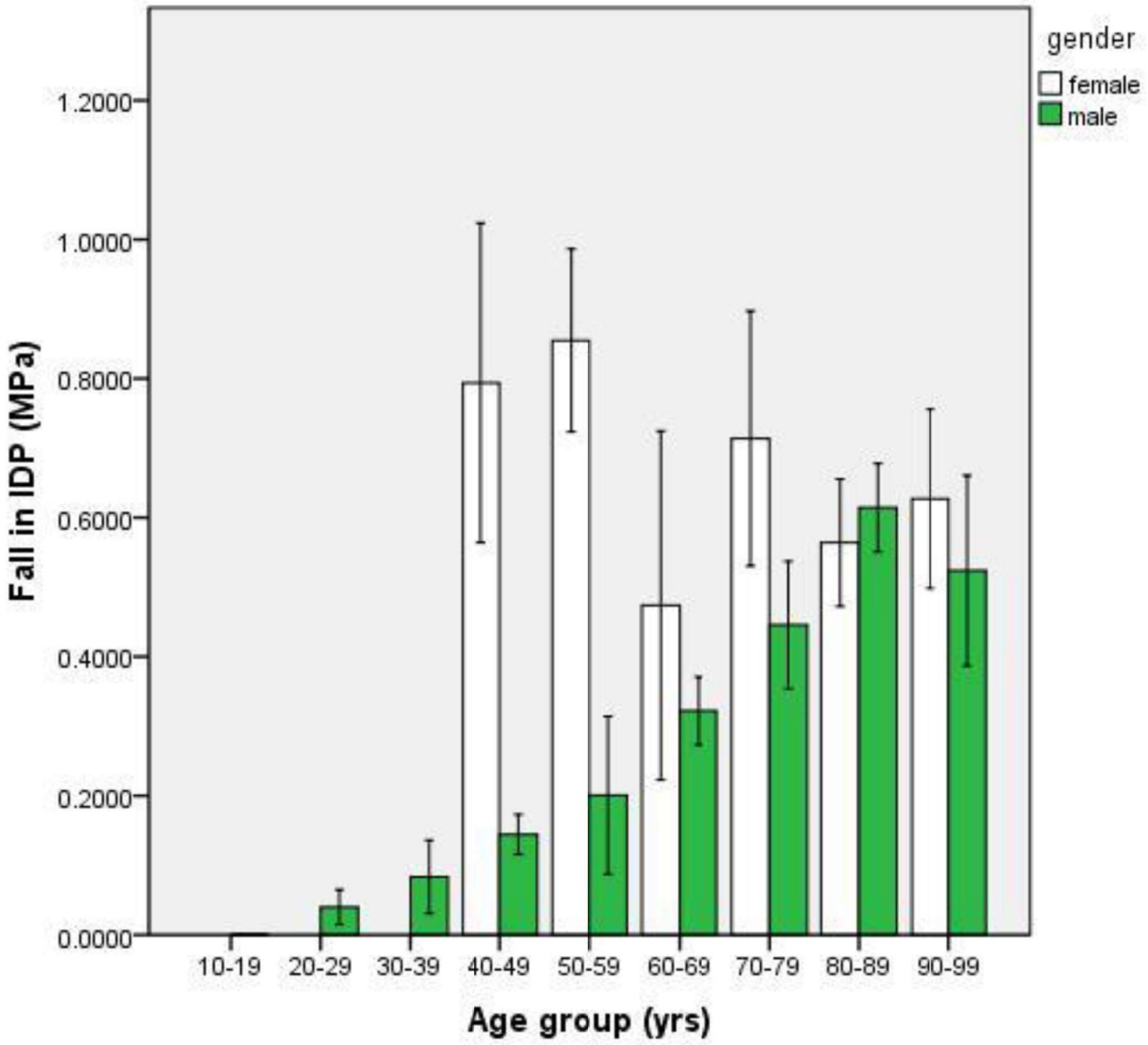
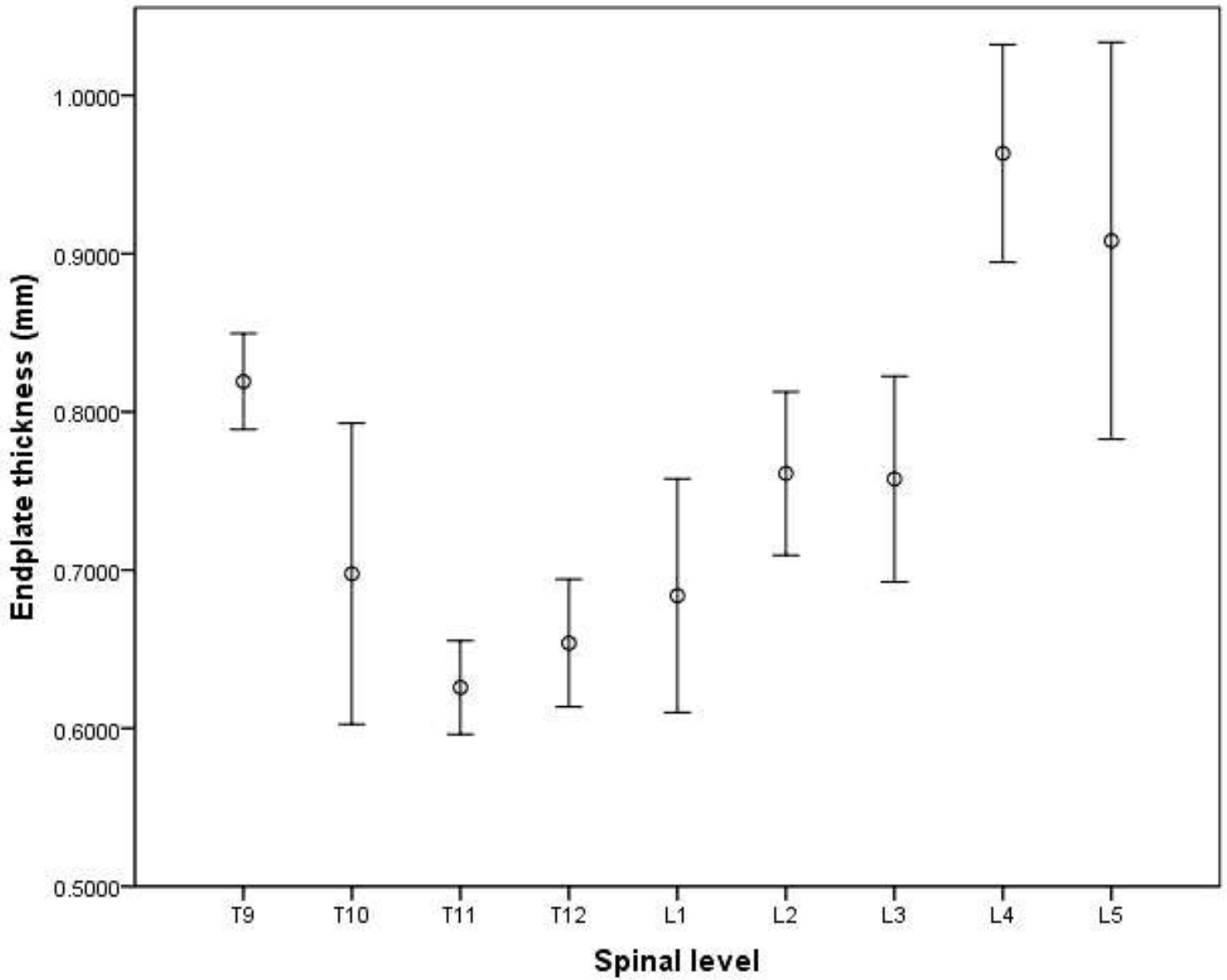

gender
female
male
1.2000
1.0000
0.8000
0.6000
0.4000
0.2000
0.0000
Fall in IDP (MPa)
10-19
20-29
30-39
40-49
50-59
60-69
70-79
80-89
90-99
Age group (yrs)

Figure 4


Endplate thickness (mm)
1.0000
0.9000
0.8000
0.7000
0.6000
0.5000
T9
T10
T11
T12
L1
L2
L3
L4
L5
Spinal level