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ANATOMY

The Effects of Age, Sex, Ethnicity, and Spinal Level on the Rate of Intervertebral Disc Degeneration

A Review of 1712 Intervertebral Discs

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Study Design. A gross anatomic and magnetic resonance imaging study of intervertebral disc (IVD) degeneration in fresh cadaveric lumbar spines.

Objective. The purpose of this study was to find the rate of IVD degeneration.

Summary of Background Data. Age, sex, race, and lumbar level are among some of the factors that play a role in IVD degeneration. The rate at which IVDs degenerate is unknown.

Methods. Complete lumbar spine segments (T11/T12 to S1) were received within 24 hours of death. The nucleus pulposus, anulus fibrosus, cartilaginous and bony endplate, and the peripheral vertebral body were assessed with magnetic resonance imaging and IVD degeneration was graded by two observers from grade 1 (nondegenerated) to grade 5 (severely degenerated) on the basis of a scale developed by Tanaka *et al*. The specimens were then sectioned and gross anatomic evaluation was performed according to Thompson *et al*.

Results. A total of 433 donors and 1712 IVDs were analyzed. There were 366 whites, 47 Africans, 16 Hispanics, 4 Asian. There were 306 male and 127 female donors. The age range was 14 to 81 years, (average: 60.5 ± 11.3). For donors greater than age 40, the L5/S1 IVD degenerated at a significantly faster rate of 0.043 per year compared to 0.031, 0.034, 0.033, 0.027 for L1/L2, L2/L3, L3/L4, L4/L5, respectively. For donors younger than 40, L5/S1 IVD degenerated at a significantly faster rate of 0.141/y compared to 0.033, 0.021, 0.031, 0.050 for L1/L2, L2/L3, L3/L4, L4/L5, respectively. Multiple regression analysis revealed that sex had no significant effect on IVD degeneration whereas African ethnicity was associated with lower Thompson score at L1/L2, L2/L3, L3/L4, L4/L5 when compared with whites.

Conclusion. The relatively early degeneration at L5–S1 in all races and lower Thompson grade in donors of African ethnicity needs further investigation. Factors such as sagittal alignment, facet joint arthritis, and genetics potentially play a role in IVD degeneration.

Key words: age, degeneration, ethnicity, intervertebral disc, rate.

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Intervertebral disc (IVD) degeneration is believed to begin as early as the second decade of life, and is viewed by most as an inevitable consequence of ageing.[1–3] The IVD comprises a centrally located nucleus pulposus (NP) core, the peripherally located multilaminar anulus fibrosus (AF), and cartilaginous endplates.

It has been shown that IVD degeneration originates in the NP, with a decrease in proteoglycan content and type II collagen synthesis, while there is an increase in denaturation of type II collagen and synthesis of type I collagen.[4–8] As the NP loses its osmotic properties, which leads to fibrosis, the NP cannot transmit forces efficiently, thus initiating the degenerative cascade that affects the AF and the whole disc.

Several authors have shown IVD degeneration to be associated with ethnicity, smoking, and vertebral level.[9–15] IVD degeneration is commonly graded by using the Thompson classification, which uses a five-category grading scheme for assessing gross morphology of midsagittal sections of human lumbar IVD.[16] Tanaka *et al* have shown a high correlation between magnetic resonance (MR) images of IVD degeneration and the Thompson grade on gross morphologic inspection with the reported κ value as high as 0.9.[17]

There are multiple reports which correlate age with disc degeneration.[1,5] However; there are no studies reporting the "rate" at which each IVD level degenerates in people with no prior history of spinal problems. Knowing the rate of IVD degeneration has an important prognostic value. Furthermore, having a noninvasive means of assessing IVD degeneration, which closely correlates with the gross morphologic grade, would further enhance the usefulness of such a diagnostic examination. The purpose of this study was to find the rate of

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IVD degeneration at each lumbar level by using MR imaging and gross macroscopic evaluation.

MATERIALS AND METHODS

Donor Population

Spine segments were harvested by and received from the Gift of Hope Organ and Tissue Donor Network after obtaining consent from the families. All the individuals had died of acute causes such as acute trauma, acute poisoning, cerebral bleeding, sepsis or myocardial infarction. None of the individuals had a history of a relevant back problem (*e.g.*, previous in-hospital treatment, surgery, or invalidity) in their medical reports (available at autopsy). Furthermore, the donors' families were questioned regarding any spine-related history. Specimens with a prior history of spine related problems were excluded from this study. Demographic data including age, sex, and ethnicity as well as the donors' cause of death were obtained. The Gift of Hope Organ and Tissue Donor Network uses an observation based three-point scale to assess donor weight. Donors are classified as underweight, normal, or overweight.

TABLE 1. Donor Demographics

Total Donors	433
Men	306
Women	127
Average age	
Men	59.13 ± 12.5
Women	61.02 ± 10.6
Age range	14–81
IVD analyzed	1712
Overweight	93
Normal weight	340
African	47
Asian	4
White	366
Hispanic	16

Donor Samples

Complete lumbar spine segments (T11/12–S1) were received within 24 hours of death of the donor. The segments included the entire spinal column as well as the surrounding muscles and ligaments. Each of the lumbar segments subsequently underwent MR imaging. Specimens that were found to have fractures, excessive osteophytes (precluding opening of the joint), or other signs of a pathologic process were excluded from this analysis. The vertebral column was then sectioned with a saw in a sagittal plane 5 mm from the midline. The NP, AF, cartilaginous and bony endplate, and the peripheral vertebral body were evaluated macroscopically by two observers.

Sample Analyses

Gross Specimen Evaluation

The NP, AF, cartilaginous and bony endplate, and the peripheral vertebral body were assessed macroscopically and IVD degeneration was graded by two observers from grade 1 (non-degenerated) to grade 5 (severely degenerated) on the basis of a scale developed by Thompson *et al*.[16]

Magnetic Resonance Image Evaluation

MR images were taken using a Siemens Magnetom Vision (Siemens AG, Munich, Germany) at 1.5 T. The operational modes employed were T1-weighted spin echo sequences (TR 500 milliseconds, TE 20 milliseconds) and T2-weighted spin echo sequences (TR 3200 milliseconds, TE 160 milliseconds). The NP, AF, cartilaginous and bony endplate, and the peripheral vertebral body were assessed by MR imaging and IVD degeneration was graded by two observers from grade 1 (normal), grade 2 (mild disc degeneration), grade 3 (moderate disc degeneration), grades 4 (severe) and 5 (severe with osteophytes > 2 mm) using a modification of the scale developed by Thompson *et al*.[16] Any discrepancy between the MR imaging-based grade and the macroscopic Thompson grade was recorded and those samples showing a different grade by these two methods were excluded from this study.

Statistical Analyses

Age, sex, and ethnicity of the donor and degeneration grade for each IVD were recorded and used for statistical analysis. Statistical analysis was performed using simple and multiple linear regression as well as simple mean comparisons using analysis of variance and Student *t* tests. Simple comparisons using student *t* test and analysis of variance were used to establish the differences between age (independent variable) and IVD degeneration in males and females, and various ethnicities. The donors were also grouped according to decades. Linear regression was used to establish the rate of IVD degeneration for each level, as well as, to analyze the differences in IVD degeneration between the various decades. The observational weight scale was not used for statistical analysis.

RESULTS

Cadaveric lumbar spine segments of 433 donors and 1712 IVDs were used for analysis. There were 366 whites, 47 blacks, 16 Hispanics, 4 Asian, among the donors of which 306 were males and 127 were females. The age range was 14 to 81 years, average age was 60.5 ± 11.3 years (Table 1).

The average Thompson grade by age and spinal level is presented in Table 2. Degeneration grade increased as one proceeded caudally down the spinal column. There was a statistically significant difference between L1/L2 and L4/L5 ($P < 0.0001$).

There was a statistically significant difference in Thompson grade observed between L2/L3 and L4/L5 ($P < 0.0001$); between L3/L4 and L4/L5 ($P < 0.0001$); between L4/L5 and L5/S1 ($P < 0.034$) and between L1/L2 and L3/L4 ($P = 0.023$).

TABLE 2. Average Thompson Grade and Age by Spinal Level

Level	Mean Thompson Grade	Mean Age	No. of IVD
L1/L2	2.79 ± 0.85	60.3 ± 11.7	401
L2/L3	2.80 ± 0.84	60.5 ± 11.5	433
L3/L4	2.86 ± 0.81	60.4 ± 11.5	435
L4/L5	2.99 ± 0.86	60.2 ± 11.7	363
L5/S1	2.93 ± 0.97	61.0 ± 11.8	80

There was a statistically significant difference between L1/2 and L4/5 ($P < 0.0001$).

There was a statistically significant difference between L2/3 and L4/5 ($P < 0.0001$).

There was a statistically significant difference between L3/4 and L4/5 ($P < 0.0001$).

There was a statistically significant difference between L4/5 and L5/S1 ($P < 0.034$).

There was a statistically significant difference between L1/2 and L3/4 ($P = 0.023$).

The average Thompson grade by decade is presented in Table 3, and the differences in Thompson grades between decades are presented in Table 4. Increase in the degeneration grade was observed with increasing age of the donor, and statistically significant differences were found between every pair of age groups compared, except for decades V and IV, II and III, III and IV, and III and V.

The average Thompson grade by spinal level and decade is presented in Table 5. There was no statistically significant difference in IVD Thompson grade between spinal levels within any decade.

Rate of Age-Related IVD Degeneration Per Spinal Level

Regression analysis revealed that age indeed predicted deterioration at all lumbar levels; however, the rate of degeneration was dependent on the level of the IVD in the spinal column (Table 6). For donors over the age of 40, the L5/S1 IVD degenerated at a significantly faster rate of 0.043 per year when compared to 0.031, 0.034, 0.033, and 0.027 for L1/L2, L2/L3, L3/L4, and L4/L5, respectively. There was no statistical difference in the rate of IVD degeneration when comparing L1/L2, L2/L3, L3/L4, and L4/L5. For donors under the age of 40, the L5/S1 IVD degenerated at a significantly faster rate of 0.141 per year when compared to 0.033, 0.021, 0.031, and 0.050 for L1/L2, L2/L3, L3/L4, and L4/L5, respectively. There was a statistically significant difference in the rate of IVD degeneration between donors under 40 years and those over 40 years at the L2/L3, L4/L5, and L5/S1 level.

Sex and IVD Degeneration Relationship for Each Spinal Level

Sex, age, and Thompson grade are presented in Table 7. The comparison in Thompson grades between males and females for each spinal level is presented in Table 8. There was no statistical difference between males and females in degeneration grades at any lumbar level ($P > 0.05$).

TABLE 3. Average Thompson Grade by Decade

Decade	N	Mean Thompson Grade	SD	SEM	Lower 95%	Upper 95%
II	4	1.38	0.91	0.45	–0.06	2.84
III	6	1.83	0.54	0.22	1.26	2.40
IV	12	2.17	0.27	0.08	1.99	2.34
V	46	2.28	0.48	0.07	2.13	2.42
VI	129	2.72	0.56	0.04	2.63	2.82
VII	142	3.00	0.65	0.05	2.90	3.11
VIII	91	3.32	0.60	0.06	3.20	3.45

Ethnicity and IVD Degeneration Relationship for Each Spinal Level

Age, ethnicity, and mean Thompson grade are presented in Table 9. Comparison between ethnicities and mean Thompson grades revealed a significant difference between whites and Africans, as shown in Table 10. There was no difference between the remaining ethnicities—white/Asian; white/Hispanic; African/Asian; African/Hispanic; Asian/Hispanic. There was a statistically significant difference between white and African ethnicities in IVD degeneration at the L2/L3 ($P = 0.021$), L3/L4 ($P = 0.006$), and L4/L5 ($P = 0.013$) levels, with African ethnicity being associated with having a lower Thompson grade. There was no difference between the ethnicities in the Thompson grades at the L1/L2 ($P = 0.0507$) and L5/S1 ($P = 0.39$) levels (Table 11).

DISCUSSION

In this study we were able to correlate IVD degeneration on the basis of MR imaging and gross morphologic examination with the age, sex, and ethnicity of the donor as well as the position of the IVD in the lumbar spine. We demonstrated a direct relationship between the age of the donor and the grade of IVD degeneration, and determined the rate of degeneration in IVDs of each level of the lumbar spine. Interestingly, the rate of degeneration per year was faster in the L5/S1 IVD relative to other lumbar segments. The difference was even more profound in the 14 to 40-age group, although the sample size was small.

One of the reasons that the rate of IVD degeneration is highest at the L5/S1 motion segment is secondary to the high magnitude of compressive forces seen at the lumbosacral junction.[18] Furthermore, anterior shear forces at the L5/S1 motion segment increase proportionally with increasing sacral angle.[18,19] Anterior and posterior disc postural loads, which are balanced at T8–T9, show the greatest difference at L5/S1.[19] IVD degeneration in the lumbar spine has been shown to be associated with motion.[17] Furthermore, it has

TABLE 4. Comparison in Thomson Grade Between Decades Using Student *t* Test

Decade (1)	Decade (2)	Difference in Mean Thomson Grade	SE Difference	Lower CL	Upper CL	*P*
VIII	II	1.94	0.30	1.34	2.53	<0.0001*
VII	II	1.62	0.30	1.03	2.21	<0.0001*
VIII	III	1.49	0.24	1.00	1.98	<0.0001*
VI	II	1.34	0.30	0.74	1.93	<0.0001*
VII	III	1.17	0.24	0.68	1.66	<0.0001*
VIII	IV	1.15	0.18	0.80	1.51	<0.0001*
VIII	V	1.04	0.10	0.83	1.25	<0.0001*
VI	III	0.89	0.24	0.40	1.38	0.0003*
V	II	0.89	0.30	0.28	1.49	0.0041*
VII	IV	0.83	0.17	0.48	1.18	<0.0001*
IV	II	0.78	0.34	0.11	1.45	0.0225*
VII	V	0.72	0.10	0.53	0.92	<0.0001*
VIII	VI	0.59	0.08	0.44	0.75	<0.0001*
VI	IV	0.55	0.17	0.20	0.90	0.0019*
VI	V	0.44	0.10	0.24	0.64	<0.0001*
V	III	0.44	0.25	–0.05	0.95	0.0830
III	II	0.44	0.38	–0.30	1.19	0.2445
IV	III	0.33	0.29	–0.24	0.91	0.2553
VIII	VII	0.31	0.08	0.16	0.47	<0.0001*
VII	VI	0.27	0.07	0.13	0.42	0.0001*
V	IV	0.10	0.19	–0.26	0.48	0.5692

**P < 0.05.*

CL indicates control limit.

TABLE 5. Average Thompson Grade by Spinal Level and Decade

Level	10–20	21–30	31–40	41–50	51–60	61–70	71–80
L1/L2	1.25 ± 0.5 (n = 4)	1.91 ± 0.66 (n = 12)	2.0 ± 0.0 (n = 11)	2.21 ± 0.55 (n = 39)	2.7 ± 0.83 (n = 115)	2.87 ± 0.80 (n = 135)	3.19 ± 0.79 (n = 86)
L2/L3	1.5 ± 1.0 (n = 4)	2.0 ± 0.42 (n = 12)	2.0 ± 0.0 (n = 11)	2.11 ± 0.48 (n = 42)	2.68 ± 0.78 (n = 127)	2.90 ± 0.80 (n = 144)	3.23 ± 0.74 (n = 94)
L3/L4	1.5 ± 1.0 (n = 4)	2.25 ± 0.62 (n = 12)	2.18 ± 0.4 (n = 11)	2.22 ± 0.60 (n = 42)	2.73 ± 0.72 (n = 127)	2.95 ± 0.76 (n = 144)	3.29 ± 0.79 (n = 93)
L4/L5	1.5 ± 1.0 (n = 4)	2.25 ± 0.75 (n = 12)	2.33 ± 0.5 (n = 9)	2.41 ± 0.68 (n = 33)	2.83 ± 0.79 (n = 112)	3.16 ± 0.80 (n = 120)	3.27 ± 0.87 (n = 72)
L5/S1	1.00 ± 1.0 (n = 1)	2.0 ± 1.41 (n = 2)	(n = 0)	2.22 ± 0.44 (n = 9)	2.56 ± 0.79 (n = 24)	3.13 ± 0.86 (n = 23)	3.54 ± 0.96 (n = 22)
	17	50	42	165	505	566	367

There was no statistically significant difference in Thompson grade between L1/L2, L2/L3, L3/L4, L4/L5, and L5/S1 for any decade.

TABLE 6. Regression Analysis of Rate of Intervertebral Disc Degeneration by Level and Age

Level	Rate of IVD Degeneration Per Year in Donors (14–40 yrs)	Rate of IVD Degeneration Per Year in Donors (41–80 yr)	No. of IVD
L1/L2	0.033 (n = 27)	0.031 (n = 404)	401
L2/L3	0.021 (n = 27)	0.034* (n = 406)	433
L3/L4	0.031 (n = 27)	0.033 (n = 405)	435
L4/L5*	0.050 (n = 25)	0.027* (n = 345)	363
L5/S1*	0.141 (n = 3)	0.043* (n = 77)	80

The difference between L4/L5 and L5/S1 was significant for both the 14- to 40-year group and the 41- to 80-year group ($P < 0.05$). The difference between the 14- to 40-year group and the 41- to 80-year group was significant at L2/L3, L4/L5, and L5/S1 ($P < 0.05$).

been shown that the proliferation of IVD cells, the formation of clefts and tears, and both mucoid and granular matrix degeneration, are statistically associated with aging and/or IVD degeneration.[3,7,8] There is a direct correlation between IVD matrix metalloproteinases expression and age, with aging IVDs containing more matrix metalloproteinases-producing cells.[3] These IVDs may therefore show a higher breakdown of collagenous matrix molecules.[3] The increased compressive loads seen at L5/S1 may trigger a more robust increase in matrix metalloproteinases leading to an increased rate of IVD degradation. Thus, we propose that it is because of all the earlier-mentioned factors that L5/S1 IVDs showed a statistically higher rate of degeneration when compared to other levels. Reporting on pooled data from three studies using the Nachemson scale, Ashton-Miller *et al* reported no difference in degeneration grades between the L3/L4, L4/L5, and L5/S1 levels.[20,21] The authors also reported that the L5/S1 disc does not degenerate before the L3/L4 disc. In contrast to the present study, the donors' clinical history was not available in the Ashton-Miller review. Therefore, no conclusions about the natural history of IVD degeneration can be made.

It would appear that there is a direct linear relationship between IVD degeneration and age after the fifth decade. Multiple regression analysis demonstrated that IVD degeneration plateaued during the third and fourth decades and then increased steadily after the fifth decade. This relationship was observed at every level. Antoniou *et al* demonstrated that synthesis of type II collagen in the NP tissues dropped

TABLE 7. Age, Sex, and Thompson Grade

Sex	N	Mean Age	SD	Mean Thompson Grade	SD
Female	127	59.13	12.5	2.81	0.70
Male	306	61.02	10.6	2.87	0.67

$P > 0.05$.

TABLE 8. Grade of Intervertebral Disc Degeneration by Sex and Level

Level	Thompson Grade in Males	Thompson Grade in Females	No. of IVD
L1/L2	2.77 ± 0.83	2.71 ± 0.82	401
L2/L3	2.81 ± 0.83	2.76 ± 0.81	433
L3/L4	2.88 ± 0.8	2.79 ± 0.82	435
L4/L5	2.99 ± 0.82	2.91 ± 0.94	363
L5/S1	2.92 ± 0.91	2.8 ± 1.10	80

with aging and increasing grade of degeneration.[5] Adult degenerative NP cells produced significantly higher amounts of proteoglycans, collagens, and aggrecan when compared with adolescent cells. These findings suggest that human NP cells are involved in repairing and replacing diminishing ECM during age-related degeneration.[5,6] It can therefore be postulated that after the fifth decade, IVD cannot repair and replace the ECM as efficiently as IVD in the third and fourth decades leading to a progressive, linear degeneration in the NP.

To the best of our knowledge this is the largest series of cadaver spines that underwent both MR imaging and gross morphologic evaluation at a single institution. It should be noted that there was no history of spinal conditions in any of the donors allowing us to report on the natural history of IVD degeneration.

There are many studies that assess IVD degeneration using MR imaging. This is a safe and reproducible way of judging not only IVD degeneration but also neural compression. MR imaging closely reflects the degenerative changes noted in the IVD on gross morphologic examination. Previously, Tanaka *et al* evaluated both MR images and cryomicrotome sections using Thompson's classifications, and found no statistical difference between the IVD degeneration grades on the basis of the two types of images.[17] However, 13 of 14 discs that were assigned to grade I based on cryomicrotome sections were assigned to grade II based on MR images. The authors concluded that MR images reflect the morphologic changes in degeneration of the IVD except for early grades I and II.

Using a five grade scale, based on T2 weighted sequence MR imaging, Boos *et al* prospectively observed 46 asymptomatic individuals during a 5-year follow-up period, and reported that IVD degeneration progressed in 17 (41.5%) of 41 patients.[13] In a cross-sectional MR imaging study of lumbar IVD degeneration in 200 healthy individuals aged 30 to 55 years, Kanayama *et al* reported that age was a significant risk factor when examining any disc level except L5/S1.[22] Over half of the subjects had moderate IVD degeneration at baseline at the L4/L5 and L5/S1 levels. Other parameters (episode of low back pain, smoking status, body mass index, and hours of standing/sitting) were not found to be significantly related to disc degeneration. In our study we noted a

TABLE 9. Ethnicity, Age, and Mean Thompson Grade

Ethnicity	N	Mean Age	SD	Mean Thompson Grade	SD	SEM	Lower 95%	Upper 95%
African	47	59.6	9.0	2.60	0.56	0.08	2.4	2.7
Asian	4	54.0	22.0	2.48	1.03	0.51	0.8	4.1
White	366	60.7	11.1	2.89	0.68	0.03	2.8	2.9
Hispanic	16	59.6	13.9	2.72	0.77	0.20	2.2	3.1

TABLE 10. Comparison Between Ethnicities and Mean Thompson Grade

Ethnicity (1)	Ethnicity (2)	Difference	SE Difference	Lower CL	Upper CL	*P*
White	Asian	0.41	0.34	–0.26	1.08	0.23
White	African	0.28	0.10	0.070	0.50	0.0094*
Hispanic	Asian	0.24	0.38	–0.51	1.00	0.52
White	Hispanic	0.16	0.18	–0.19	0.53	0.37
African	Asian	0.12	0.35	–0.57	0.82	0.72
Hispanic	African	0.12	0.20	–0.29	0.53	0.56

P > 0.05 for age.

CL indicates control limit.

significant correlation between IVD degeneration and age at every level, with L5/S1 demonstrating the highest rates and grades of IVD degeneration.

Smoking data were not available in this analysis, however, some authors reported that smoking has not been shown to have an effect on the prevalence of IVD degeneration.[13,22] Other authors found both smoking, and obesity as detrimental factors for lumbar IVD degeneration.[9–12] All of these studies evaluated subjects in a narrow age range. Battie *et al* used MR imaging to study pairs of identical twins highly discordant for cigarette smoking, and reported 18% greater mean IVD degeneration scores in the lumbar spines of smokers than nonsmokers.[9] Liuke *et al*, also studied the association between being overweight and having lumbar IVD degeneration in 129 working middle-aged men and showed that body mass index of 25 or more increased the risk of lumbar IVD degeneration.[12]

TABLE 11. Grade of Intervertebral Disc Degeneration by Ethnicity and Level

Level	Thompson Grade in Whites	Thompson Grade in Africans
L1/L2*	2.80 ± 0.82 (n = 334)	2.54 ± 0.84 (n = 42)
L2/L3*	2.84 ± 0.84 (n = 362)	2.54 ± 0.70 (n = 46)
L3/L4*	2.91 ± 0.80 (n = 361)	2.56 ± 0.73 (n = 46)
L4/L5*	3.01 ± 0.86 (n = 303)	2.65 ± 0.62 (n = 38)
L5/S1	2.98 ± 0.97 (n = 63)	2.50 ± 0.70 (n = 13)

**Statistically significant difference (P < 0.05).*

These authors excluded L1/L2 and L5/S1 from their analysis and used one examiner to review all the available images.

One of the drawbacks of this study was related to the harvesting technique, which often sectioned the spine through the L5/S1 disc space reducing the amount of L5/S1 IVDs available for analysis to 80. This was especially evident when samples were analyzed by decade and there were no L5/S1 specimens in the 31- to 40-age range available for analysis.

In this study, IVD degeneration was influenced by ethnicity at L1/L2, L2/L3, L3/L4, and L4/L5 levels, but not at the L5/S1 level. Although IVD degeneration is multifactorial, genetics certainly plays a role.[23] The Tt and the tt genotypes of Taq I polymorphism of the vitamin D receptor gene have been associated with IVD degeneration.[14,23,24] This association was shown in a Finnish study examining 85 pairs of monozygotic twins between the ages of 39 and 69 years.[24] In a Japanese study examining 205 Japanese volunteers and patients between the ages of 20 and 29 years, the Tt genotype was more frequently associated with multilevel disc disease, severe disc degeneration, and disc herniation than the TT genotype.[14] The frequency of this "risk t-allele" is significantly different between the three major ethnic populations, and is found in 8% of Asians, 31% of Africans, and 43% in whites.[25] Gruber *et al* demonstrated a relationship between ethnicity and IVD cell proliferation, with advanced age and Middle Eastern ethnicity resulting in poorer proliferative ability of cultured cells.[15] We suspect that the lack of difference in grades of IVD degeneration between ethnicities at the L5/S1 level is a result of the high forces that this motion segment sees in all ethnic groups. The higher frequency of the "risk t-allele" in whites may partially explain the higher IVD grades seen in this population in our study.

CONCLUSIONS

The rate of IVD degeneration in donors with no history of spine problems was highest at L5/S1, most likely due to the high forces seen at this motion segment. The relatively early degeneration at L5/S1 in all ethnicities needs further investigation. Other factors such as sagittal alignment, facet joint arthritis, and genetic predisposition play a role in IVD degeneration.

➢ Key Points

- ❑ The rate of IVD degeneration was highest at L5/S1.
- ❑ The rate of IVD degeneration was higher at L5/S1 for donors younger than 40 then those older than 40.
- ❑ Donors of African ethnicity had a lower rate of IVD degeneration when compared with whites.

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