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## **Disc Versus Vertebral Body Contribution to Lumbar Lordosis in Asymptomatic**

### **Subjects**

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This study was approved by regional ethics committees (approval N° 6001 and 6061 C.P.P. Ile-de France VI and FM 312 ethical committee at the Saint-Joseph University, Beirut). All participants provided their informed written consent.

**Conflicts of interest:**

The authors have no conflict of interest to declare relatively to this study.

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**ABSTRACT**

**Study design:** Retrospective study of a multicentric prospective database.

**Objective:** This study aimed at describing the relative contribution of vertebral bodies versus discs to lumbar lordosis, and its variation with age and pelvic incidence.

**Summary of Background Data:** While studies sought to determine the physiological magnitude and distribution of lumbar lordosis, data regarding its anatomical composition is lacking.

**Methods:** This study included healthy volunteers with full-body stereoradiographs in free-standing position, without lumbosacral transitional vertebra or age under 18. The following parameters were analyzed: age, sex, pelvic incidence (PI), lumbar lordosis (LL). Posterior heights and sagittal Cobb angles between upper and lower endplate for each lumbar disc and each vertebral body were measured from L1 to S1. Ratios of contribution to LL were calculated for each disc and vertebral body. The cohort was divided into four age groups and four PI groups.

**Results:** 645 subjects were included, mean age was  $37.6 \pm 16.3$ , 51% of females. There was a significant decrease in total lumbar disc lordosis with age ( $-48.9 \pm 9.7^\circ$  to  $-42.9 \pm 10.2^\circ$ ), occurring in lower LL. Vertebral bodies were significantly more kyphotic in Seniors than Youngs ( $-8.9 \pm 8.4^\circ$  versus  $-5.0 \pm 9.4^\circ$ ,  $p=0.03$ ), driven by a significant increase in kyphosis of L1 and L2 bodies. Vertebral body contribution to LL significantly increased between groups as PI increased, from a median of 8.0% to 20.5% ( $p<0.001$ ). This decrease in disc contribution in favor of vertebral bodies mainly took place in lower LL.

## **Conclusion**

This study highlights the importance of vertebral contribution to lumbar lordosis, ranging from 8 to 21% among PI groups. Lumbar lordosis decreased with aging through decreased disc lordosis in the lower lumbar spine and increased body kyphosis in the upper lumbar

spine. These results may help surgeons in the assessment of sagittal alignment and the selection of operative technique to achieve surgical correction.

## **KEYPOINTS**

- There was a significant decrease in total lumbar disc lordosis with aging, occurring in lower lumbar lordosis.
- Vertebral bodies were significantly more kyphotic in elderly than young subjects.
- Lower lumbar discs presented similar lordosis among PI groups, while upper LL discs and all vertebral bodies were more lordotic with PI increase.
- Vertebral body contribution to lumbar lordosis significantly increased between groups as PI increased, from a median of 8.0% to 20.5%.
- These results may help surgeons in the assessment of sagittal alignment and the selection of operative technique to achieve surgical correction.

## INTRODUCTION

Bipedalism and erect posture are unique features of hominids, illustrating Darwin's theory on the biological evolution of species<sup>1</sup>. These major changes were made possible by anatomical changes in pelvic anatomy and spinal curvatures<sup>2</sup>. Human pelvises are shorter and broader than chimpanzee's, with more ventrally curved iliac blades<sup>2,3</sup>. Lumbar lordosis (LL) is a key parameter to maintain standing posture<sup>4</sup>, and a major driver in sagittal malalignment. Therefore, LL has been extensively studied in the literature in terms of magnitude and distribution<sup>5</sup>. Its close relationship with pelvic parameters has been demonstrated<sup>6</sup>, and the PI (pelvic incidence)-LL mismatch was introduced as a parameter to define lumbar malalignment<sup>7</sup>. More recently, the importance of lumbar curvature distribution between proximal and distal lordosis has become a mainstay to avoid mechanical complications<sup>8</sup>.

While studies sought to determine the physiological magnitude and distribution of lumbar lordosis<sup>9</sup>, data regarding its anatomical composition is lacking. Indeed, there might be a common belief that lumbar lordosis is associated with the discs while the vertebral body shape is often overlooked. Such misconception could lead to fallacious sagittal alignment assessment and operative planning. In a comparative study, Been et al. reported that lumbar vertebrae in humans were more lordotic than those of macaques<sup>10</sup>, corroborating the anatomical changes to function evolution, accounting for 11% of lumbar lordosis<sup>11</sup>. Shefi et al. studied LL anatomy variation with growth, showing LL increase through an augmentation in disc wedging<sup>12</sup>. Nevertheless, some questions regarding the anatomical components of lumbar lordosis remained unanswered: How important is the vertebral body contribution to LL? Is it affected by age? Is it affected by pelvic incidence?

In an attempt to better understand lumbar lordosis and its variations, this study aimed to describe the relative contribution of vertebral bodies versus discs to lumbar lordosis, and its variation with age and pelvic incidence.

## **MATERIAL AND METHODS**

### ***Population***

This retrospective study of a prospectively collected multicentric database included healthy volunteers with full-body EOS® stereoradiographs in free-standing position, with no previous spine surgery, no major pain in spine, hip or knee, no spinal deformity, nor spondylolisthesis. Participants were enrolled in four centers between 2011 and 2023. Subjects with lumbosacral transitional vertebrae or aged under 18 years old were not included. This study was conducted after IRB approval (CPP Ile-de-France VI – Approval #6001 and 6061). All participants gave their informed consent to participate in this study.

### ***Parameters***

Demographic parameters included age and sex. Radiographic parameters were obtained after 3D reconstruction of the spine and pelvis using a previously validated semi-automated method<sup>13</sup>, which demonstrated good reproducibility<sup>14</sup>. First, the spinal line was drawn from C3 to L5 on the frontal and lateral views. The software then generated a 3D spinal reconstruction and retro-projected the 3D models of the vertebrae on the radiographs. This

model was then manually adjusted to precisely fit vertebral contours visible on the radiographs.

All the 3D reconstructions were performed by the same trained physician (orthopedic spine surgeon) ensuring radiographic measurement consistency. The following radiographic parameters (**Figure 1**) were computed:

- Lumbar lordosis (LL, measured from L1 upper endplate to S1 endplate)
- Upper LL (ULL, measured from L1 upper endplate to L4 upper endplate)
- Lower LL (LLL, measured from L4 upper endplate to S1 endplate)
- Sagittal Cobb angles between upper and lower endplates for each lumbar disc and each vertebral body
- Pelvic incidence (PI)
- Disc and vertebral body posterior height, measured from the posterior corner of the vertebral bodies

Ratios of contribution to LL were calculated for each disc and vertebral body, and contributions to Upper or Lower LL respectively. By convention, lordosis is expressed as negative values and kyphosis as positive ones.

The following clinical scores were assessed: Oswestry disability index (ODI), lumbar visual analog scale (L-VAS), the mental and physical components of short form-12 (SF12-MCS and SF12-PCS, respectively).

### ***Statistical analyses***

First, the demographics and radiographic parameters for the whole cohort were described using mean  $\pm$  standard deviation (SD). The contribution of each disc and vertebral body angles to LL was described as median and inter-quartile range (IQR). The statistical analysis was carried out using RStudio (Version 2023.09.1+494; from R software Version 4.3.0 –

packages prettyR, vioplot, gmodels), with p-values lower than 0.05 considered significant. Correlations were sought between lumbar lordosis components (vertebral bodies, discs) and age and PI, using Spearman's correlation tests.

The cohort was divided into four age groups: "Young" (18-30 years), "Adult" (30-50 years), "Middle-aged" (50-65 years) and "Senior" (65+ years). Demographics and radiographic data were described in each group. The segmental angles and relative contributions of discs and bodies were compared between groups using ANOVAs for normally distributed variables or Kruskal-Wallis tests otherwise. Post-hoc analyses were performed using pairwise t-tests or Wilcoxon's tests for each pair of groups, with Bonferroni correction.

For PI effect analysis, four PI subsets were determined using Jenks discretization: "Low" (<40°), "Average low" (40-50°), "Average high" (50-60°), and "High" (>60°). The same analyses as for age groups were carried out according to PI groups.

## RESULTS

### *Cohort*

Out of the 879 subjects in the database, 645 were eligible for this study (**Figure 2**), of whom 329 were females (51%). The mean age was 37.6±16.3 years (range: 18-90), the mean LL was -56.9±11.3°, and the mean PI was 49.2±9.5°. The mean segmental lordosis for each disc and vertebral body is given in **Table 1**.

Overall, the median disc contribution to LL was 85.9% (IQR:77.4 – 95.4), and the median vertebral body contribution was 14.1% (IQR: 4.6 – 22.6). Segmental details are reported in **Table 2**. The median disc contribution to upper LL was 115.0% (IQR: 98.2 – 139.8) compensating kyphotic vertebral bodies with a median contribution to upper LL at -15.0% (IQR: -39.8 - 1.8). PI was not correlated with lower LL discs but with upper LL discs and total disc lordosis (both  $r=-0.3$ ,  $p<0.001$ ), total vertebral body lordosis ( $r=-0.4$ ,  $p<0.001$ ),

and both ULL and LLL vertebral bodies (both  $r=-0.3$ ,  $p<0.001$ ). Age showed low correlations with LL discs ( $r=0.1$ ,  $p=0.009$ ), ULL bodies ( $r=0.1$ ,  $p<0.001$ ), and total body lordosis ( $r=0.1$ ,  $p<0.001$ ).

### ***Age groups***

The segmental Cobb angles for each lumbar disc and vertebral body among age groups are reported in **Table 1**. Youngs' mean PI was significantly lower than that of Adults and Middle-Aged subjects ( $p=0.02$  and  $0.03$  respectively). There was a significant decrease in total lumbar disc lordosis between Adults, Middle-aged, and Seniors ( $p=0.01$  and  $0.001$ , respectively), occurring in lower LL (**Figure 3**). Vertebral bodies were significantly more kyphotic in Seniors than in Young subjects ( $p=0.03$ ) (**Figure 3**).

Total disc contribution to LL tended to increase with age ( $p=0.13$ ), led by a significant increase in L1-L2 disc contribution (**Table 2**). Total vertebral body contribution to LL tended to decrease with age ( $p=0.13$ ), driven by a significant increase in kyphosis of L1 and L2 bodies (**Table 2**). Although statistically significant, the variations in disc posterior heights between age groups were minor (Supplementary Appendix 1, Supplemental Digital Content 1, <http://links.lww.com/BRS/C518>). Total body posterior height decreased with age ( $p=0.02$ ).

### ***Pelvic incidence groups***

The four PI groups had comparable ages ( $p=0.51$ ). The segmental Cobb angles for each lumbar disc and vertebral body among PI groups are reported in **Table 3**. L1-S1 lordosis significantly increased between every group of increasing PI ( $p<0.001$ ), through disc and body lordosis increase (**Table 3**). Lower LL discs presented similar lordosis among PI groups, while upper LL discs and all vertebral bodies were more lordotic with PI increase (*all*  $p<0.001$ ).

Vertebral body contribution to LL significantly increased between groups as PI increased, from a median of 8.0% to 20.5% ( $p < 0.001$ ) (**Figure 4**). The decrease in disc contribution mainly took place in lower LL: median L5-S1 contribution was 26.9% in Low- versus 18.4% in High-PI group ( $p < 0.001$ ) (**Table 4**). Conversely, the contribution of vertebral bodies in upper LL increased much more sharply with PI than in lower LL (Supplementary Appendix 2, Supplemental Digital Content 2, <http://links.lww.com/BRS/C519>). The detailed contribution of each disc and vertebral body is provided in **Table 4**. Posterior height of vertebral bodies decreased with PI augmentation (**Table 5**).

## DISCUSSION

Understanding how lumbar lordosis is distributed among vertebral bodies and discs across different age and PI groups is essential for restoring normal alignment in patients. However, the vertebral body contribution to lumbar lordosis is often overlooked. Data regarding the anatomical constitution of lumbar lordosis remains scarce<sup>11</sup>, although it is crucial to delineate its components to define surgical correction accurately. This study aimed to challenge the “square prejudice” and highlighted significant findings to enhance our understanding of lumbar lordosis and its variations with age and PI. On average, 14% of lumbar lordosis originates from the vertebral bodies. Older subjects exhibited less lordotic discs in the lower LL and more kyphotic vertebral bodies in upper LL. With increasing PI, the relative contribution of vertebral bodies to LL increased significantly.

This study demonstrated that age significantly impacts the contribution of vertebral bodies and discs to lumbar lordosis, with a more pronounced variation from Middle-Aged to Senior groups. Aging was associated with decreased disc lordosis, primarily occurring in the

lower LL. Specifically, the contribution of L4-L5 disc to LL decreased in the elderly, while the L1-L2 disc lordosis increased, likely due to a compensatory mechanism <sup>15</sup>. This finding aligns with clinical observation of a higher prevalence of degenerative discs in the lower lumbar spine <sup>16</sup>. This loss of lordosis can drive sagittal malalignment, which is compensated by hyperextension in the upper LL discs.

Aging was also associated with more kyphotic vertebral bodies, mainly found at L1 and L2, with increased kyphotic values and negative contribution to lordosis in the elderly. This phenomenon may be explained by the increasing kyphotic curvature in the thoracic spine and lumbar discs with aging <sup>17</sup>, which places greater constraints on the anterior aspect of the vertebral body, making it more likely to remodel in case of poor bone density <sup>18</sup>.

Interestingly, pelvic incidence was strongly associated with vertebral body morphology in the lumbar spine. Lumbar lordosis increased with PI through both increased disc and vertebral lordosis. All lumbar vertebral bodies were more lordotic in high-PI subjects. The vertebral contribution to LL is essential as it shifted from 8% in low-PI subjects to 21% in high-PI group, primarily in the lower LL. These results are in line with Been et al. who described increased vertebral wedge angle in human lordotic lumbar spines compared to macaque spines <sup>10</sup>.

Posterior height of vertebral bodies decreased with higher PI, indicating that bodies in high-PI subjects are smaller as this more lordotic shape is not due to an increased anterior wall but a shorter posterior wall. This finding suggests that curvature drives vertebral body shape, possibly due to inhibited posterior wall growth because of greater posterior mechanical loads, supporting the Hueter-Volkman law <sup>19</sup>. The increase in lumbar lordosis with PI did not involve lower lumbar discs, which presented similar lordosis values across all PI group.

This concurs with Pesenti et al.'s conclusion that pelvic incidence was only correlated with proximal LL <sup>5</sup>.

The results of this study challenge the conventional understanding of vertebral body shape in the lumbar spine, with various implications. First, these findings could help adapt spinal correction by considering vertebral body lordosis. For instance, implanting interbody cages in the upper LL spine, without accounting for bony lordosis could lead to LL overcorrection, increasing the risk of proximal junctional failure by shifting the upper body's center of mass posteriorly <sup>20,21</sup>. Second, these results offer a better understanding of the lumbar lordosis anatomical composition and its variation with age and PI. Lastly, the data provided in this study are relevant for designing spinal models used in research or educational purposes <sup>22</sup>.

### ***Limitations***

This study presents several limitations. First, its cross-sectional nature limits the assessment of the actual aging effect on disc and body lordosis, restricting comparison to different age groups. Moreover, no MRI nor CT-scans were used in this study. MRI would have allowed further assessment of the disc, and CT-scans could have estimated the association between the kyphotic change in vertebral bodies and bone density. Last, subjects' height and weight were not analyzed, potentially biasing the interpretation of posterior disc and vertebral body heights and lumbar alignment.

### **CONCLUSION**

This study highlights the importance of vertebral contribution to lumbar lordosis, averaging 14% and increasing to 21% in the high-PI group. Lumbar lordosis decreased with aging through decreased disc lordosis in the lower lumbar spine and increased body kyphosis in the upper lumbar spine. These results may help surgeons assessing sagittal alignment and selecting operative techniques to achieve surgical correction.

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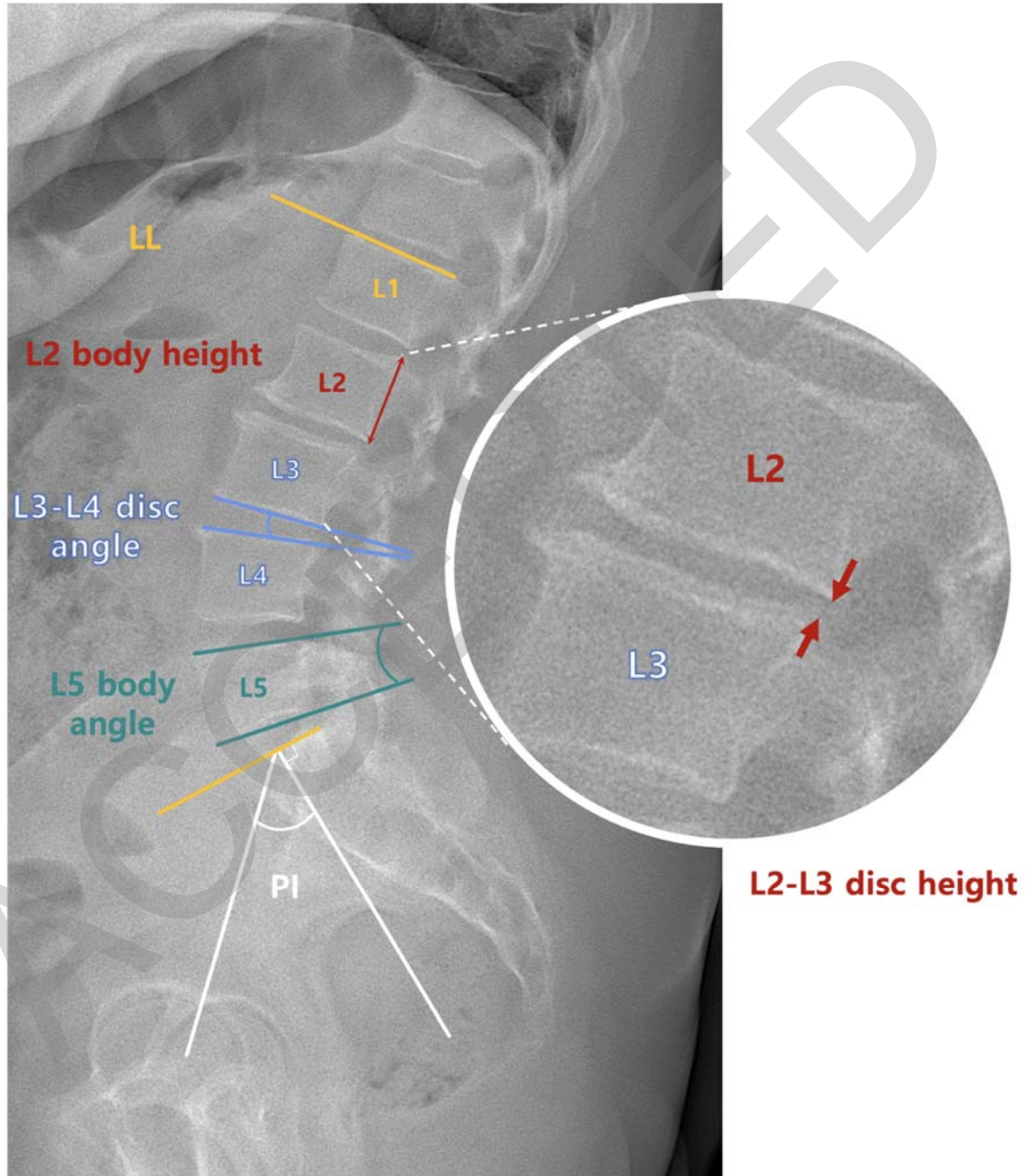
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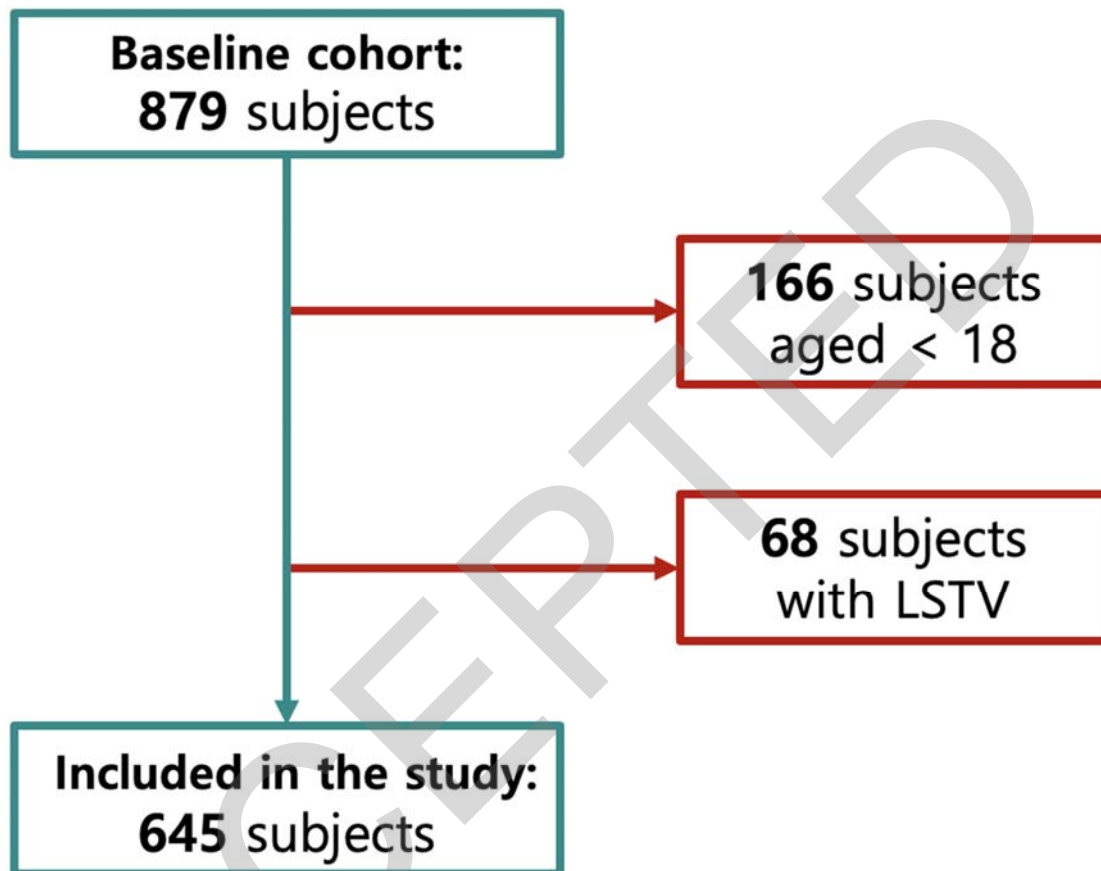
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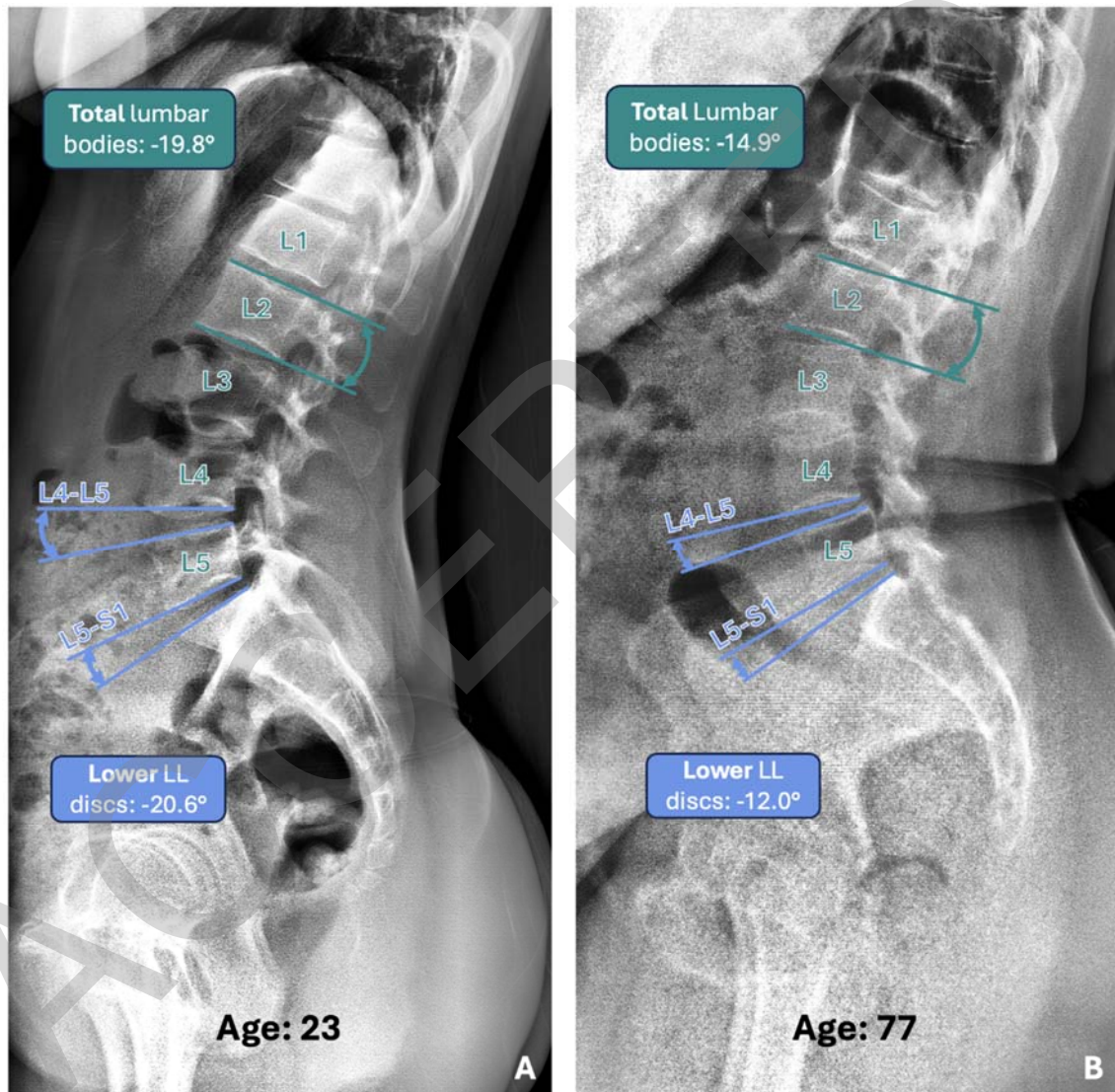
**Figure 1:** Measurement of pelvic incidence (PI), L1-S1 lumbar lordosis (LL), sagittal Cobb angle of L5 vertebral body and L3-L4 disc, as well as the posterior height for L2 body and L2-L3 disc.



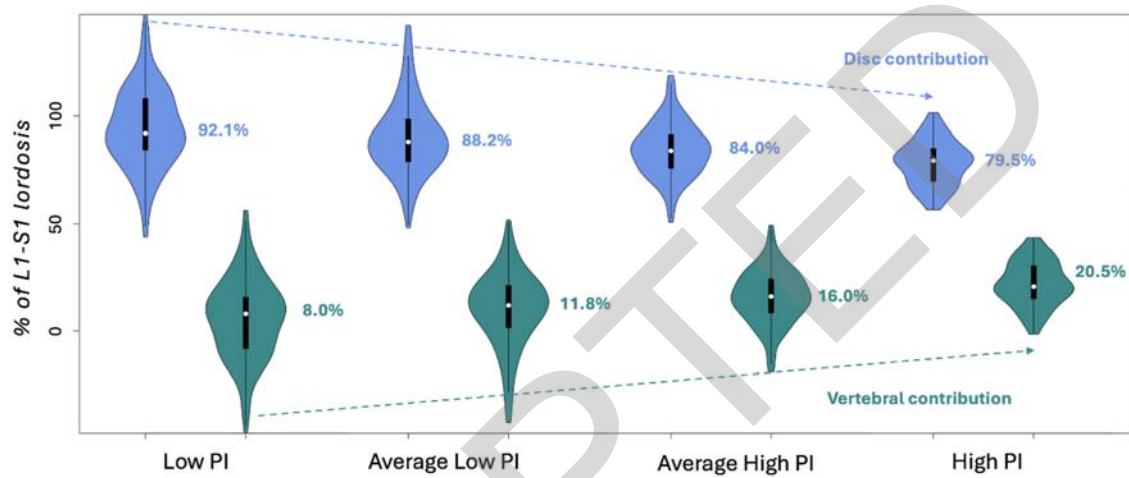
**Figure 2:** Flow-chart. LSTV: lumbosacral transitional vertebra.



**Figure 3:** Example of two subjects aged 23 (A) and 77 (B) years old, showing significantly greater lower LL disc lordosis (L4-L5 and L5-S1) and total lumbar bodies lordosis (L1 to L5 bodies) in subject A.



**Figure 4:** Violin plots representing disc and vertebral body contribution to LL in each of the PI groups. Median contributions to LL are marked aside the violins.



<i>Age groups</i>	<i>Young</i>	<i>Adult</i>	<i>Middle-Aged</i>	<i>Senior</i>	<i>p-value</i>	<i>Cohort</i>
<b><i>n</i></b>	291	193	110	51	-	645
<b><i>Age (y.)</i></b>	23.7±2.7	38.3±5.8	57.3±4.7	71.7±5.7	-	37.6±16.3
<b><i>PI (°)</i></b>	47.9±9.4	50.5±9.6	50.6±9.6	49.0±9.0	0.008*	49.2±9.5
<b><i>Disc angles (°)</i></b>						
<b><i>L1-L2 disc</i></b>	-5.4±3.0	-5.6±3.0	-6.0±3.2	-6.3±3.1	0.13	-5.6±3.1
<b><i>L2-L3 disc</i></b>	-8.2±2.7	-8.5±2.9	-8.3±2.9	-7.7±3.4	0.30	-8.3±2.9
<b><i>L3-L4 disc</i></b>	-10.1±2.7	-10.6±2.9	-9.9±3.4	-9.4±3.6	0.11	-10.1±3.0
<b><i>L4-L5 disc</i></b>	-12.2±3.4	-12.3±3.7	-10.5±3.7	-8.8±3.7	<0.001*	-11.7±3.7
<b><i>L5-S1 disc</i></b>	-13.1±5.7	-14.1±6.2	-14.2±5.9	-10.8±6.1	0.001*	-13.4±6.0
<b><i>Total disc</i></b>	-48.9±9.7	-51.1±10.8	-48.9±11.2	-42.9±10.2	<0.001*	-49.1±10.5
<b><i>Vertebral body angles (°)</i></b>						
<b><i>L1 body</i></b>	3.2±2.6	3.6±2.7	3.7±2.7	4.3±2.6	0.03*	3.5±2.7
<b><i>L2 body</i></b>	0.8±2.5	1.5±2.3	1.3±2.7	2.3±2.7	<0.001*	1.2±2.5
<b><i>L3 body</i></b>	-1.2±2.4	-0.9±2.5	-1.0±2.8	-0.5±2.9	0.36	-1.0±2.6
<b><i>L4 body</i></b>	-2.6±2.8	-2.5±2.6	-2.8±3.1	-3.1±3.8	0.57	-2.7±2.9
<b><i>L5 body</i></b>	-9.0±3.1	-9.1±3.6	-8.0±3.5	-8.0±3.5	0.02*	-8.8±3.4
<b><i>Total body</i></b>	-8.9±8.4	-7.4±9.2	-6.8±9.6	-5.0±9.4	0.01*	-7.8±9.0
<b><i>Regional and total lordosis (°)</i></b>						
<b><i>Upper LL</i></b>	-20.9±8.0	-20.6±8.1	-20.2±8.5	-17.3±10.4	0.04*	-20.4±8.4
<b><i>Lower LL</i></b>	-36.9±6.7	-37.9±7.1	-35.5±6.6	-30.6±8.7	<0.001*	-36.5±7.2
<b><i>L1-S1</i></b>	-57.8±10.5	-58.5±11.4	-55.7±10.4	-47.9±13.0	<0.001*	-56.9±11.3
<b><i>Clinical scores</i></b>						
<b><i>ODI</i></b>	2.1 ± 4.3	2.5 ± 5.5	4.3 ± 6.9	3.1 ± 6.3	0.39	2.8 ± 5.6
<b><i>L-VAS</i></b>	0.7 ± 1.3	1.2 ± 1.7	1.6 ± 2.0	1.2 ± 2.2	0.005*	1.1 ± 1.7
<b><i>SF12-PCS</i></b>	54.7 ± 5.5	54.4 ± 5.9	51.8 ± 6.9	49.6 ± 7.4	<0.001*	53.7 ± 6.2
<b><i>SF12-MCS</i></b>	51.1 ± 8.8	51.9 ± 7.1	51.7 ± 8.4	55.3 ± 5.3	0.20	51.7 ± 8.1

**Table 1:** Mean±SD segmental Cobb angles for each lumbar disc and vertebral body and clinical scores among age groups. P-values from the ANOVA or Kruskal-Wallis analyses are marked with an asterisk if <0.05.

<i>Age groups</i>	<i>Young</i>	<i>Adult</i>	<i>Middle-Aged</i>	<i>Senior</i>	<i>K-W</i>	<i>Cohort</i>
<b>Disc contribution to L1-S1 lordosis (%)</b>						
<i>L1-L2 disc</i>	<b>9.4</b> (6.6–12.8)	<b>9.6</b> (6.5–12.7)	<b>10.9</b> (7.9–14.1)	<b>14.1</b> (10.5–17.0)	<i>&lt;0.001*</i>	<b>9.9</b> (6.8–13.5)
<i>L2-L3 disc</i>	<b>14.0</b> (11.4–17.2)	<b>14.3</b> (12.0–17.1)	<b>15.0</b> (11.8–17.6)	<b>16.2</b> (11.9–18.9)	<i>0.21</i>	<b>14.3</b> (11.6–17.4)
<i>L3-L4 disc</i>	<b>17.5</b> (14.9–20.5)	<b>18.1</b> (15.3–21.1)	<b>17.1</b> (14.5–21.4)	<b>19.4</b> (14.7–25.3)	<i>0.14</i>	<b>17.8</b> (14.9–21.1)
<i>L4-L5 disc</i>	<b>20.9</b> (17.2–24.9)	<b>21.0</b> (18.2–24.7)	<b>18.8</b> (15.2–23.1)	<b>17.2</b> (11.8–25.5)	<i>0.002*</i>	<b>20.5</b> (16.6–24.7)
<i>L5-S1 disc</i>	<b>23.1</b> (16.9–30.1)	<b>24.5</b> (18.0–31.8)	<b>26.1</b> (18.0–33.3)	<b>22.4</b> (14.3–29.8)	<i>0.08</i>	<b>23.7</b> (17.1–31.2)
<i>Total disc</i>	<b>85.1</b> (76.5–93.1)	<b>86.8</b> (78.0–97.1)	<b>86.8</b> (78.2–98.2)	<b>88.7</b> (78.2–99.6)	<i>0.13</i>	<b>85.9</b> (77.4–95.4)
<b>Vertebral body contribution to L1-S1 lordosis (%)</b>						
<i>L1 body</i>	<b>-5.1</b> (-8.7–-2.3)	<b>-5.5</b> (-9.5–-3.0)	<b>-6.3</b> (-10.7–-2.9)	<b>-9.2</b> (-12.8–-5.4)	<i>0.001*</i>	<b>-5.8</b> (-9.5–-2.8)
<i>L2 body</i>	<b>-1.2</b> (-4.1–-1.6)	<b>-2.0</b> (-5.2–-0.2)	<b>-2.4</b> (-5.2–-0.6)	<b>-3.1</b> (-9.0–-0.9)	<i>&lt;0.001*</i>	<b>-1.8</b> (-5.0–0.8)
<i>L3 body</i>	<b>2.1</b> (-0.8–4.4)	<b>1.9</b> (-1.3–3.9)	<b>1.1</b> (-1.8–4.6)	<b>2.0</b> (-3.3–4.1)	<i>0.41</i>	<b>2.0</b> (-1.3–4.2)
<i>L4 body</i>	<b>4.4</b> (1.4–7.5)	<b>4.3</b> (1.5–7.4)	<b>4.3</b> (1.8–7.9)	<b>5.7</b> (2.4–12.3)	<i>0.14</i>	<b>4.4</b> (1.5–7.7)
<i>L5 body</i>	<b>15.3</b> (11.7–18.8)	<b>15.5</b> (12.0–19.1)	<b>14.4</b> (9.9–19.7)	<b>17.2</b> (12.3–22.1)	<i>0.28</i>	<b>15.4</b> (11.5–19.2)
<i>Total body</i>	<b>14.9</b> (6.9–23.5)	<b>13.2</b> (2.9–22.0)	<b>13.2</b> (1.8–21.8)	<b>11.4</b> (0.4–21.8)	<i>0.13</i>	<b>14.1</b> (4.6–22.6)
<b>Regional lordosis contribution to L1-S1 lordosis (%)</b>						
<i>Upper LL</i>	<b>36.3</b> (29.4–41.8)	<b>34.8</b> (28.4–41.4)	<b>36.3</b> (28.5–42.5)	<b>37.5</b> (27.0–47.4)	<i>0.51</i>	<b>36.1</b> (29.0–42.3)
<i>Lower LL</i>	<b>63.7</b> (58.2–70.6)	<b>65.2</b> (58.6–71.6)	<b>63.7</b> (57.5–71.5)	<b>62.5</b> (52.6–73.0)	<i>0.51</i>	<b>63.9</b> (57.7–71.0)

**Table 2:** Median (IQR) contribution to LL for each lumbar disc and vertebral body among age groups. P-values from the Kruskal-Wallis (K-W) analyses are marked with an asterisk if  $<0.05$ .

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<b>PI groups</b>	<b>Low</b>	<b>Average low</b>	<b>Average high</b>	<b>High</b>	<b>p-value</b>
<b>n</b>	99	253	212	81	-
<b>PI (°)</b>	35.1 ± 3.6	45.3 ± 2.9	54.2 ± 2.9	65.6 ± 5.8	-
<b>Age (years)</b>	36.4 ± 17.4	37.2 ± 16.5	38.3 ± 16.1	38.6 ± 14.9	0.51
<b>Disc angles (°)</b>					
<b>L1-L2 disc</b>	-4.1 ± 2.9	-5.3 ± 3.2	-6.5 ± 2.8	-6.2 ± 2.7	<0.001*
<b>L2-L3 disc</b>	-6.9 ± 2.7	-8.1 ± 3.1	-8.8 ± 2.5	-9.0 ± 2.6	<0.001*
<b>L3-L4 disc</b>	-8.8 ± 2.7	-10.0 ± 2.9	-10.5 ± 2.9	-11.4 ± 3.1	<0.001*
<b>L4-L5 disc</b>	-10.9 ± 3.2	-11.6 ± 3.7	-11.8 ± 3.5	-12.4 ± 4.5	0.05
<b>L5-S1 disc</b>	-12.1 ± 5.6	-13.9 ± 6.0	-13.2 ± 5.9	-13.6 ± 6.5	0.08
<b>Total disc</b>	-42.9 ± 10.4	-49.0 ± 10.2	-50.7 ± 10.2	-52.6 ± 9.4	<0.001*
<b>Vertebral body angles (°)</b>					
<b>L1 body</b>	4.7 ± 2.8	3.7 ± 2.8	3.0 ± 2.4	2.4 ± 2.3	<0.001*
<b>L2 body</b>	2.3 ± 2.7	1.6 ± 2.5	0.8 ± 2.2	-0.5 ± 2.2	<0.001*
<b>L3 body</b>	-0.2 ± 2.4	-0.8 ± 2.8	-1.3 ± 2.3	-2.0 ± 2.4	<0.001*
<b>L4 body</b>	-1.8 ± 2.5	-2.2 ± 3.1	-2.9 ± 2.5	-4.3 ± 2.9	<0.001*
<b>L5 body</b>	-7.9 ± 3.3	-8.2 ± 3.3	-9.1 ± 3.0	-10.6 ± 4.1	<0.001*
<b>Total body</b>	-2.9 ± 8.2	-6.0 ± 9.2	-9.5 ± 7.4	-14.9 ± 7.7	<0.001*
<b>Regional and total lordosis (°)</b>					
<b>Upper LL</b>	-13.0 ± 6.9	-19.0 ± 7.4	-23.2 ± 7.0	-26.7 ± 8.3	<0.001*
<b>Lower LL</b>	-32.8 ± 7.0	-36.0 ± 7.0	-37.0 ± 6.5	-40.9 ± 7.5	<0.001*
<b>L1-S1</b>	-45.8 ± 9.6	-55.0 ± 9.5	-60.2 ± 9.0	-67.8 ± 10.4	<0.001*
<b>Clinical scores</b>					
<b>ODI</b>	1.2 ± 2.8	3.7 ± 6.4	2.7 ± 6.0	2.4 ± 3.5	0.004*
<b>L-VAS</b>	0.6 ± 1.3	1.3 ± 1.8	1.2 ± 1.8	0.9 ± 1.1	0.04*
<b>SF12-PCS</b>	54.1 ± 4.7	54.0 ± 6.3	53.0 ± 7.2	54.0 ± 5.0	0.65
<b>SF12-MCS</b>	53.5 ± 5.0	51.1 ± 9.5	51.9 ± 7.6	51.2 ± 7.4	0.51

**Table 3:** Mean ± SD segmental Cobb angles for each lumbar disc and vertebral body and clinical scores among PI groups. P-values from the ANOVA or Kruskal-Wallis analyses are marked with an asterisk if <0.05.

<i>PI groups</i>	<i>Low</i>	<i>Average low</i>	<i>Average high</i>	<i>High</i>	<i>K-W</i>
<b><i>Disc contribution to L1-S1 lordosis (%)</i></b>					
<b><i>L1-L2 disc (%)</i></b>	<b>8.6</b> (4.1 – 12.6)	<b>9.5</b> (5.8 – 13.9)	<b>10.5</b> (8.3 – 13.6)	<b>8.5</b> (6.6 – 11.8)	<i>0.005*</i>
<b><i>L2-L3 disc (%)</i></b>	<b>15.4</b> (11.3 – 18.5)	<b>14.6</b> (11.4 – 17.8)	<b>14.3</b> (12.2 – 17.2)	<b>13.0</b> (11.1 – 16.2)	<i>0.02*</i>
<b><i>L3-L4 disc (%)</i></b>	<b>19.4</b> (16.1 – 23.1)	<b>18.1</b> (14.7 – 21.5)	<b>16.9</b> (14.9 – 20.4)	<b>16.9</b> (14.7 – 19.3)	<i>0.003*</i>
<b><i>L4-L5 disc (%)</i></b>	<b>23.1</b> (19.6 – 29.4)	<b>21.1</b> (17.0 – 25.1)	<b>19.4</b> (16.2 – 23.0)	<b>18.7</b> (14.3 – 23.1)	<i>&lt;0.001*</i>
<b><i>L5-S1 disc (%)</i></b>	<b>26.9</b> (19.3 – 33.2)	<b>26.0</b> (19.2 – 32.6)	<b>21.9</b> (16.1 – 28.0)	<b>18.4</b> (13.6 – 28.2)	<i>&lt;0.001*</i>
<b><i>Total disc (%)</i></b>	<b>92.1</b> (84.4 – 108.2)	<b>88.2</b> (78.9 – 98.6)	<b>84.0</b> (75.9 – 91.7)	<b>79.5</b> (69.8 – 85.1)	<i>&lt;0.001*</i>
<b><i>Vertebral body contribution to L1-S1 lordosis (%)</i></b>					
<b><i>L1 body (%)</i></b>	<b>-10.7</b> (-14.6 – -5.9)	<b>-6.6</b> (-10.2 – -3.6)	<b>-4.8</b> (-8.1 – -2.2)	<b>-3.6</b> (-5.3 – -1.9)	<i>&lt;0.001*</i>
<b><i>L2 body (%)</i></b>	<b>-4.2</b> (-9.3 – -1.4)	<b>-2.4</b> (-5.5 – 0.3)	<b>-1.4</b> (-3.7 – 0.9)	<b>0.7</b> (-1.0 – 2.2)	<i>&lt;0.001*</i>
<b><i>L3 body (%)</i></b>	<b>-0.3</b> (-2.9 – 3.4)	<b>2.1</b> (-2.0 – 4.3)	<b>2.0</b> (-0.2 – 4.2)	<b>2.6</b> (0.2 – 4.7)	<i>&lt;0.001*</i>
<b><i>L4 body (%)</i></b>	<b>3.6</b> (-0.4 – 6.9)	<b>3.6</b> (0.8 – 7.8)	<b>4.6</b> (2.1 – 7.7)	<b>6.3</b> (4.3 – 8.3)	<i>&lt;0.001*</i>
<b><i>L5 body (%)</i></b>	<b>18.1</b> (11.7 – 22.5)	<b>14.7</b> (11.4 – 18.6)	<b>15.3</b> (11.7 – 18.7)	<b>15.8</b> (11.8 – 19.5)	<i>0.008*</i>
<b><i>Total body (%)</i></b>	<b>8.0</b> (-8.2 – 15.6)	<b>11.8</b> (1.4 – 21.1)	<b>16.0</b> (8.3 – 24.1)	<b>20.5</b> (15.0 – 30.2)	<i>&lt;0.001*</i>
<b><i>Regional lordosis contribution to L1-S1 lordosis (%)</i></b>					
<b>Upper LL</b> (% of LL)	<b>29.7</b> (19.6 – 36.1)	<b>34.6</b> (27.1 – 41.4)	<b>38.1</b> (33.9 – 43.6)	<b>39.8</b> (34.3 – 44.5)	<i>&lt;0.001*</i>
<b>Lower LL</b> (% of LL)	<b>70.3</b> (64.0 – 80.4)	<b>65.4</b> (58.6 – 72.9)	<b>61.9</b> (56.5 – 66.1)	<b>60.2</b> (55.5 – 65.7)	<i>&lt;0.001*</i>

**Table 4:** Median (IQR) segmental Cobb angles for each lumbar disc and vertebral body among PI groups. P-values from Kruskal-Wallis analyses are marked with an asterisk if <0.05.

<i>PI groups</i>	<i>Low</i>	<i>Average low</i>	<i>Average high</i>	<i>High</i>	<i>p-value</i>
<i>n</i>	99	253	212	81	-
<b><i>Lumbar discs posterior height (mm)</i></b>					
<i>L1-L2 disc</i>	6.4 ± 1.3	6.3 ± 1.3	6.3 ± 1.2	6.8 ± 1.4	0.01*
<i>L2-L3 disc</i>	6.7 ± 1.2	6.5 ± 1.9	6.5 ± 1.3	7.0 ± 1.3	0.006*
<i>L3-L4 disc</i>	6.9 ± 1.4	6.7 ± 1.4	6.5 ± 1.3	6.7 ± 1.4	0.11
<i>L4-L5 disc</i>	6.1 ± 1.4	5.9 ± 1.4	5.7 ± 1.5	5.6 ± 1.5	0.05
<i>L5-S1 disc</i>	5.2 ± 2.2	4.9 ± 1.9	4.9 ± 2.0	4.9 ± 1.6	0.74
<i>Total disc</i>	31.2 ± 4.2	30.3 ± 4.4	29.9 ± 4.3	31.0 ± 4.2	0.02*
<b><i>Lumbar vertebral bodies posterior height (mm)</i></b>					
<i>L1 body</i>	27.7 ± 2.1	26.9 ± 2.3	26.6 ± 2.1	26.3 ± 1.8	<0.001*
<i>L2 body</i>	28.0 ± 2.3	27.3 ± 2.0	27.0 ± 2.2	26.3 ± 2.1	<0.001*
<i>L3 body</i>	27.5 ± 2.1	27.0 ± 2.2	26.8 ± 2.3	26.3 ± 2.0	0.002*
<i>L4 body</i>	27.1 ± 2.2	26.5 ± 2.3	26.4 ± 2.2	25.7 ± 2.4	<0.001*
<i>L5 body</i>	25.5 ± 2.2	25.2 ± 2.2	25.0 ± 2.0	24.4 ± 2.3	0.008*
<i>Total body</i>	135.8 ± 8.5	132.9 ± 8.8	131.8 ± 9.0	129.0 ± 8.5	<0.001*

**Table 5:** Mean ± SD posterior height for each lumbar disc and vertebral body among PI groups. P-values from the ANOVA or Kruskal-Wallis analyses are marked with an asterisk if <0.05.