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<https://doi.org/10.1007/s00586-020-06526-0>

Breen, A., Mellor, F., Morris, A. and Breen, A., 2020. An in-vivo study exploring correlations between early-to-moderate disc degeneration and flexion mobility in the lumbar spine. *European Spine Journal*, 29 (10), 2619-2627.

Available online: <https://doi.org/10.1007/s00586-020-06526-0>

1 **An in vivo study exploring correlations between early-to-moderate disc degeneration and**
2 **flexion mobility in the lumbar spine**

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4
5 **Abstract**

6 Purpose: Early disc degeneration (DD) has been thought to be associated with loss of spine
7 stability. However, before this can be understood in relation to back pain, it is necessary to
8 know the relationship between DD and intervertebral motion in people without pain. This
9 study aimed to find out if early to moderate DD is associated with intervertebral motion in
10 people without back pain.

11 Methods: Ten pain free adults, aged 51-71 received recumbent and weight bearing MRI
12 scans and quantitative fluoroscopy (QF) screenings during recumbent and upright lumbar
13 flexion. Forty individual level and 10 composite (L2-S1) radiographic and MRI DD gradings
14 were recorded and correlated with intervertebral flexion ROM, translation, laxity, and
15 motion sharing inequality and variability for both positions.

16 Results: Kinematic values were similar to previous control studies. DD was evidenced up to
17 moderate levels by both radiographic and MRI grading. Disc height loss correlated slightly,
18 but negatively with flexion during weight bearing flexion ($R=-0.356$, $p=0.025$). Composite
19 MRI DD and T2 signal loss evidenced similar relationships ($R= -0.305$, $R= -0.267$) but did not
20 reach statistical significance ($p=0.056$, $p=0.096$). No significant relationships between any
21 other kinematic variables and DD were found.

22 Conclusion: This study found only small, indefinite associations between early-to-moderate
23 DD and intervertebral motion in healthy controls. Motion sharing in the absence of pain
24 was also not related to early DD, consistent with previous control studies. Further research
25 is needed to investigate these relationships in patients.

Key words: back pain, disc degeneration, instability, imaging

26 Introduction

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2 27 The role of disc degeneration (DD) in the biomechanics of chronic back pain has been
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5 28 unclear for many years and important questions remain outstanding. For example, it has
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7 29 long been theorised that early DD is associated with 'dysfunction', that progression is
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10 30 followed by an 'unstable' phase, and advanced degeneration brings 'stabilisation' [1].
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12 31 However, providing evidence for this, let alone any association with pain, has proved
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15 32 difficult. Studies using flexion-extension radiographs and later MR imaging have failed to
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17 33 confirm an association between DD and abnormal movement [2, 3] , probably due to an
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20 34 inability to provide accurate and reliable measurement of subtle intervertebral motion, let
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23 35 alone correlate it with DD *in-vivo* [4].
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26 36 A number of studies using quantitative fluoroscopy (QF) have investigated continuous multi-
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28 37 segmental lumbar intervertebral motion in detail, finding quantifiable differences in motion
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31 38 patterns between patients with back pain and controls [5, 6], [7-9]. One considered patients
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34 39 with and without DD, finding more out of plane motion in the latter [10] while another
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36 40 found substantial correlations between DD and the degree of unequal motion sharing (MSI)
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39 41 during recumbent passive flexion in patients, but not in controls [8] (Figure 1). Thus, DD is
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42 42 implicated not only in *in vivo* interactions between levels, but also in the back pain
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44 43 experience. This study also compared weight bearing active flexion in patients with
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46 44 controls, and found that the variability of motion sharing (MSV) was substantially correlated
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49 45 with DD in patients, but not controls [8] (Figure1).
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52 46 In a further study of CNSLBP patients and controls, individual level weight bearing MSV was
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55 47 found to be greater at L4-5 in patients than controls, while L5-S1 received significantly less
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58 48 and L2-3 more of the overall motion in patients [11]. Yet another QF study included the
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3 49 measurement of mid-range attainment rate, (or laxity) at each level from L2-S1 and found it
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6 50 not to be higher in patients CNSLBP than normative reference limits [9].
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9 51 Given these complexities, it is difficult to understand the role of DD in CNSLBP, or to
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11 52 evaluate the Kirkaldy-Willis and Farfan hypothesis [1]. To approach this in CNSLBP patients,
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13 53 we must first determine the presence or absence of associations between DD and
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15 54 intervertebral motion in people without pain. The aims of the present study were
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17 55 therefore to find out if weight bearing or recumbent MSI and MSV, flexion ROM, translation
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19 56 or laxity are associated with DD in pain free controls with early to moderate DD.
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21 22 57 **Methods**

23 24 25 58 ***Participants***

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28 59 Ten healthy participants aged between 51 and 71 years with no history of disabling back
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30 60 pain over the previous year were recruited from a group of pain free volunteers who were
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32 61 participating in an ongoing normative QF study of recumbent and weight bearing
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34 62 intervertebral flexion motion. Following imaging, those who were found to have at least
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36 63 one intervertebral level with DD of at least Grade 2 on the Kellgren and Lawrence scale were
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38 64 invited to also have recumbent and weight bearing MRI scans [12]. Participant age, sex,
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40 65 height and weight were recorded.
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50 51 67 ***Imaging***

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54 68 Fluoroscopic sequences were obtained using a Siemens Arcadis Avantic C-arm fluoroscope
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56 69 (Siemens GMBH, Germany), recording at 15fps during controlled lumbar flexion motion
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59 70 through 40° in the lateral decubitus position and 60° in the standing position. For
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71 recumbent screening, participants lay on a movable table whose trunk section was
72 motorised and driven by a controller (Atlas Clinical Ltd). For standing imaging, they stood
73 with their right side against an upright motion frame with their pelvises secured and
74 followed a motorised arm rest which guided their flexion motion. The controllers
75 accelerated at 6° s^{-2} for the first second followed by a uniform 6° s^{-2} thereafter. Following
76 screening, the images were inspected by the authors (AB and FM) and all participants with
77 at least one level with DD of at least Grade 2 also received supine and semi-recumbent
78 sitting MRI scans on the same day. These were obtained using a Paramed MR Open 0.5T
79 scanner (Paramed ASG, Italy). Patients received supine and recumbent sitting T2 sagittal
80 and axial scans from L2-S1 (Fast Spin Echo, Matrix 256x208, Slice thickness 5mm, Gap 1mm)

81 ***Image analysis***

82 All images were inspected for incidental findings by a consultant radiologist (AM) who also
83 performed the DD grading. The fluoroscopic sequences were exported to a computer
84 workstation and analysed using manual image registration of the first image and thereafter
85 bespoke frame to frame tracking codes written in Matlab (2013 – The Mathworks Ltd
86 Cambridge). Anonymised image sequences were analysed by one operator (FM) and
87 outputted to an Excel spreadsheet in the form of frame to frame intervertebral angular
88 rotations throughout each motion sequence. The displacements between pairs of vertebrae
89 were calculated using Distortion Compensated Radiographic Analysis, which is based on
90 landmarks identified on the vertebral body ‘corners’ and provides measurement of
91 translation independent of the position of the centre of rotation [13]. Accuracy and
92 repeatability for intervertebral rotations, translation and laxity using this method have been

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3 94 intervertebral motion outputs [8, 17, 18].
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6 95 Figure 1 about here
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9 96 ***Assessment of disc degeneration***
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12 97 Radiographic DD was graded 0-4 for each level by a consultant radiologist (AM) from the
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15 98 initial sagittal fluoroscopic image [12]. This gave a composite measure of structural change
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18 99 in the form of reduced disc height, osteophytes, bone sclerosis and deformation, giving a
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20 100 maximum composite score of 16 for the 4 intervertebral levels[8, 19].
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23 101 For MRI DD the Jarosz Atlas scale was used, employing radiologist visual assessment of both
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26 102 supine and weight bearing scans (Appendix) [20]. This unpublished 6-section tool has 5
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29 103 four-point semi-quantitative scales (0-3) consisting of disc height loss, T2-weighted disc
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31 104 signal intensity loss, disc extension into the spinal canal, endplate marrow changes and
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34 105 osteophytosis. There is a further scale for alignment, scored 0 or 1, giving a maximum score
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36 106 per level of 16, or a maximum composite score of 64 for all 4 levels from L2-S1. In addition
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39 107 to the overall scores, disc height loss and signal intensity loss were included as subscales in
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41 108 the analysis in the absence of explicit measures of these common MRI DD variables.
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45 109 ***Ethics***
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48 110 All participants gave written informed consent to their involvement in the study, which
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51 111 received ethical approval from the UK South West 3 Research Ethics Committee (REC
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53 112 Reference 10/H0106/65).
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115 **Statistical analysis**

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3 116 Descriptive analysis was performed for all variables, including their values during weight
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6 117 bearing and recumbent imaging. Following inspection for normality (Shapiro Wilk test), the
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9 118 mean and maximum flexion ROM, translation, laxity MSI and MSV scores were calculated
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11 119 for both recumbent and weight bearing lumbar flexion and the results compared with each
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14 120 other (2-tailed Wilcoxon test with 5% significance) and with an existing normative dataset
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16 121 [21]. Weight bearing and recumbent radiographic and MRI DD scores were also compared.
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19 122 Nonparametric bivariate correlations (Spearman rank correlation) were calculated for both
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22 123 recumbent and weight bearing flexion ROM, translation and laxity against their respective
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25 124 segmental DD gradings. Finally, MSI and MSV in both orientations were correlated against
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27 125 their composite DD scores for all participants. To interpret the relevance of the correlations
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30 126 obtained, an 'R' score of >0.80 was considered 'excellent', >0.60-0.80 'substantial', 0.40-0.60
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32 127 'moderate' and <0.40 'slight' [22]. All data were analysed using Stats Direct statistical
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35 128 software (V2.07.0008, Birkenhead).

38 129 **Results**

41 130 Complete data were obtained for all participants, whose personal characteristics and
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44 131 kinematic and DD scores are shown in Table 1. The population was mainly female, average
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47 132 age 61, with normal BMI. The 5 kinematic variables showed similar average scores to a
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49 133 previous normative database study [21] and flexion ROM and MSV gave significantly larger
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52 134 scores on weight bearing than on recumbent QF recordings ($p \leq 0.01$). Participants received
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55 135 a mean (SD) equivalent radiation dose of 0.399 mSv (0.149) from fluoroscopy, which is 0.901
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57 136 mSv less than a standard radiographic investigation of the lumbar spine.

137 Table 1 about here

138 DD always scored higher when assessed from weight bearing examinations, but this only
139 reached statistical significance for composite MRI and disc height loss ($p < 0.04$). However,
140 DD was generally at the lower end of the DD ranges registering between 25% and 58% of
141 their maximum possible scores on weight bearing assessment (lowest, radiographic DD 25%,
142 highest, weight bearing disc height loss 58%), indicating that this population represented
143 early to moderate DD. In order to optimise the range of relationships between kinematics
144 and DD, all correlations reported here were taken using weight bearing DD assessments
145 (Table 2).

146 Table 2 about here

147 There were no significant correlations between upright or recumbent MSI or MSV and any
148 kind of DD, although a substantial negative correlation between recumbent MSI and
149 radiographic DD approached significance ($R = -0.610$, $p = 0.06$) (Table 3). Overall, this is
150 consistent with previous studies in pain free controls [8, 9].

151 Table 3 about here

152 There were slight negative linear correlations between disc height loss and flexion ROM ($R = -$
153 0.356 , $p = 0.025$) and between MRI DD and flexion ROM ($R = -0.305$, $p = 0.056$), with
154 assessments of both motion and DD performed weight bearing. (Figure 2 a, b). However,
155 the latter did not quite reach significance. Scatterplots of all correlations between weight
156 bearing DD assessments and both recumbent and weight bearing kinematic variables are
157 shown in the Supplementary Material.

158 Figure 2 a,b about here

159 **Discussion**

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3 160 This study found a slight negative correlation between disc height reduction on MRI and
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6 161 flexion ROM only, as assessed during weight bearing lumbar flexion in healthy controls
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9 162 without back pain and with early to moderate DD. No correlations were found with
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11 163 translation or laxity or with any kinematic variable during passive recumbent motion. This
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14 164 finding is, if anything, the reverse of the relationship proposed by Kirkaldy-Willis and Farfan
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16 165 [1]. Weight bearing MRI scans returned a significantly greater loss of disc height than
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19 166 recumbent ones, which is consistent with a previous study that found that positional
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21 167 changes tend to be more frequent on weight bearing MRI scans [23]. However, a similar
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24 168 weak negative correlation was also found between flexion ROM and disc height loss on the
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26 169 recumbent MRI scans ($R=-0.350$, $p=0.027$).

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30 170 No other significant associations were found between DD and intervertebral motion values,
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32 171 which were comparable to other normative studies [21]. This suggests, (but does not
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35 172 prove) that it may have been the motion abnormalities in the symptomatic patient studies,
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37 173 rather than the degenerative changes, that were the main drivers of nociceptive pain [8]. A
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40 174 review of post-fusion adjacent segment kinematic studies, where DD was implicated,
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43 175 concluded that there appears to be no overall kinematic changes at the rostral or caudal
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45 176 levels adjacent to a fusion [24]. Furthermore, although the levels of DD recorded in this
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48 177 study have been associated with the 'dysfunctional', or at most, 'unstable' phase of DD, the
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50 178 flexion ROMs found here were similar to other studies of pain free participants [21].

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54 179 However, cadaveric studies have found associations between neutral zone (NZ) length and
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56 180 radiographic DD [25], although these have also given contradictory results, where one found

181 radiographic and MRI DD to be associated with decreased flexion ROM [26], while another
182 found small increases in the NZ with MRI, but not with radiographic DD [27].

183 There are inaccuracies inherent in radiographic studies of intervertebral motion *in vivo*
184 which can be largely overcome by radiostereometric analysis (RSA). However, the
185 invasiveness of this method makes it unsuitable for use in asymptomatic controls, as well as
186 largely inaccessible for patients with chronic, nonspecific low back pain (CNLBP) [28].
187 Nevertheless, one RSA study of 18 patients with back pain being considered for lumbar
188 fusion did not detect an 'instability' phase in early DD [29].

189 The natural successor to the present biomechanical study would be a cross-sectional clinical
190 study along the same lines. A previous study that compared patients with treatment-
191 resistant back pain to controls (but did not assess DD) also found the composite value of
192 MSI to be higher in patients than controls [9]. However, laxity, translation and flexion ROM
193 were not greater in patients. Therefore, a repeat of the present study with a larger
194 population and a DD assessment similar to the present one (i.e. using recumbent and weight
195 bearing QF and MRI) could tell us whether the MSI marker is linked to DD *in vivo*.

196 If similar results are found at individual levels in CNLBP patients, (i.e. little or no association
197 between DD and IV motion), it will be evidence of an absence of direct DD involvement in
198 the pain process, consistent with the findings of Axelsson and Karlsson [29]. However, if DD
199 is again associated with increased MSI in recumbent examinations and is also associated
200 with individual level weight bearing intervertebral flexion motion sharing changes as
201 recently found in patients, this would provide evidence that the disc is not usually the
202 nociceptive source [8, 9]. The task then would be to add the assessment of other known

203 pain generators (e.g. muscle hypoxia, loading stresses, fatiguability) to such investigations to
204 determine the prevalence of these as nociceptive stimuli.

205 If we consider the relevance of the destabilisation-restabilisation theory [1] in respect of
206 patients, it is not unexpected that there would be little relationship between intervertebral
207 motion and DD in healthy controls, as this is consistent with the lack of correlation with
208 radiographic DD found in other studies and its contrast with the strong correlations ($R=0.70$
209 and 0.85) with MSI and MSV found in patients with CNSLBP [8]. What is yet to be
210 determined is whether MSI and/or MSV are related to the MRI DD factors in patients and
211 whether flexion ROM, translation or laxity are involved. One possible correlate with the
212 kinematic variables presented here is disc shear stiffness, which is becoming assessable *in*
213 *vivo* by MR elastography [30]. Other forms of imaging, such as diffusion weighted MRI could
214 also be explored as it may allow more complex associations with kinematics to be assessed
215 in patients [31]. However, continuous *in vivo* dynamic motion assessments will be required
216 as opposed to plain radiographs or kMRI, which only records categorical motion data from
217 quasi-static measurements [32, 33].

218 Also to be considered is the siting, as well as the severity of degenerated discs when
219 attempting to explore associations between DD, intervertebral motion and CNSLBP. Recent
220 QF research comparing CNSLBP patients to controls in terms of individual level motion
221 sharing throughout upright flexion suggests that in patients, L5-S1 receives less and L2-3
222 more of the motion [11]. Given the apparent importance of motion sharing in symptoms,
223 and in the light of the present study, it would be useful to investigate the influence of DD
224 graded using upright MRI, on these motion distributions and their relationships to disability.
225 This may help to explain the findings of Cheung et al, who reported that pain and disability

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226 was greater in patients with 'continuous level DD' than 'skipped level DD' [34]. This has
227 been further pursued by von Forrell et al, using FE modelling and finding higher
228 intervertebral stresses to be associated with continuous level DD compared with skipped
229 level DD [35]. Prospective studies of kinematics and stresses are becoming accessible using
230 QF motion analysis along with FE models based on 3-D MRI scans and which could include
231 muscle demands [36]. Future studies of patients might therefore consider the distribution
232 of degenerate discs along with their grades, kinematics and loading stresses in relation to
233 such muscle demands and disability.

234 **Limitations**

235 The present study was limited by small participant numbers (n=10), which reduced the
236 power to find significant correlations between MSI, MSV and composite DD. However, the
237 number of individual intervertebral level DD measures and kinematic markers was 40, and
238 should have revealed true correlations if not affected by level-specific differences in DD.

239 Indeed, a biomechanical study by Roussouly and Pinheiro-Franco proposed that there are
240 level-specific patterns in DD [37]. However, a later study by Torrie et al found that lumbar
241 spinal subtype, based on morphology, was not statistically significantly correlated with DD
242 [38] For the composite measures of MSI, MSV and composite radiographic disc
243 degeneration however, a larger sample will be necessary to investigate significant
244 relationships.

245 This study was also confined to nonparametric linear regression analysis, by virtue of the
246 categorical nature of the DD gradings, while the kinematic data were interval in nature. This
247 prevented the detection of any nonlinear associations throughout the spectrum of DD

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3 248 severity. More advanced methods for imaging the disc quantitatively and objectively could
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6 249 remove these problems.

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12 250 **Conclusion**

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14 251 In an older pain free population with early to moderate DD, this study found only small,
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16 252 indefinite associations with intervertebral mobility. Furthermore, only small and negative
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18 253 correlations were found between weight bearing flexion ROM and disc height loss, which is
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20 254 not consistent with the Kirkaldy-Willis and Farfan hypothesis [1]. No significant correlations
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22 255 were found between any other measure of DD and flexion ROM, translation, laxity, MSI or
23
24 256 MSV. These relationships may be different in patients with CNSLBP.

25 257 **Acknowledgements**

26
27
28 258 This study received funding from The Radiological Research Trust and Paramed ASG.

29
30 259 The MRI Disc Degeneration Atlas was reproduced by kind permission of Frances MK

31
32 260 Williams PhD FRCP(E), Professor of Genomic Epidemiology, Hon Consultant Rheumatologist

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371 List of Figures

372 Figure captions

373 Figure 1.

374 Short title: Derivation of MSI and MSV

375 Descriptive caption: Example of the measurement of continuous proportional intervertebral

376 motion during the flexion and return motion cycle of 4 intervertebral levels. Changes in

377 angle between adjacent vertebrae are measured throughout the motion cycle (a) and are

378 converted into proportional intervertebral contributions to the motion of the L2-S1 spine

379 (b). The ranges of the proportional intervertebral contributions are calculated (c). Motion

380 share inequality (MSI) was established as the mean of all the ranges throughout the flexion

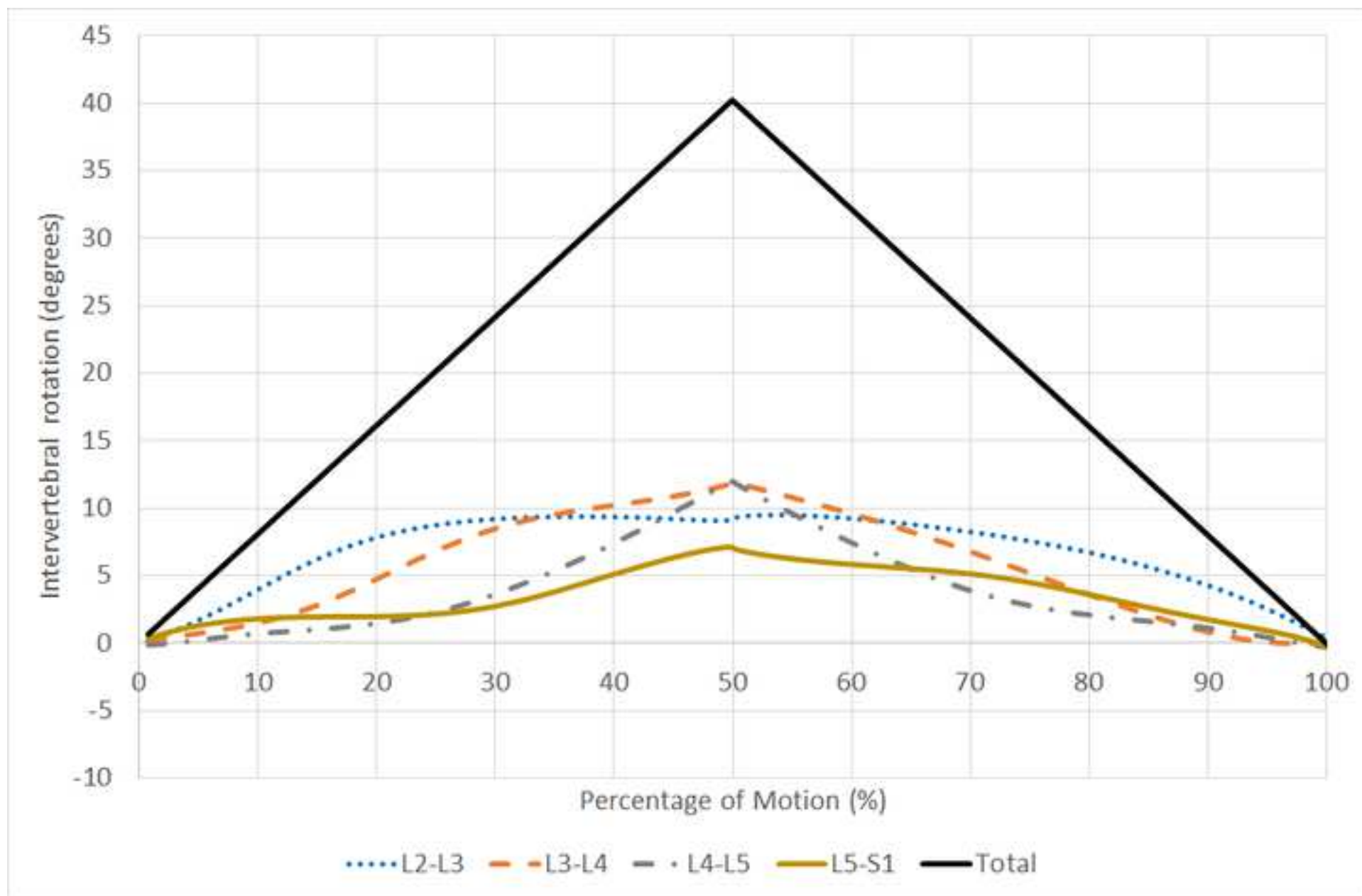
381 and return bend and motion share variability (MSV) was the standard deviation of this range

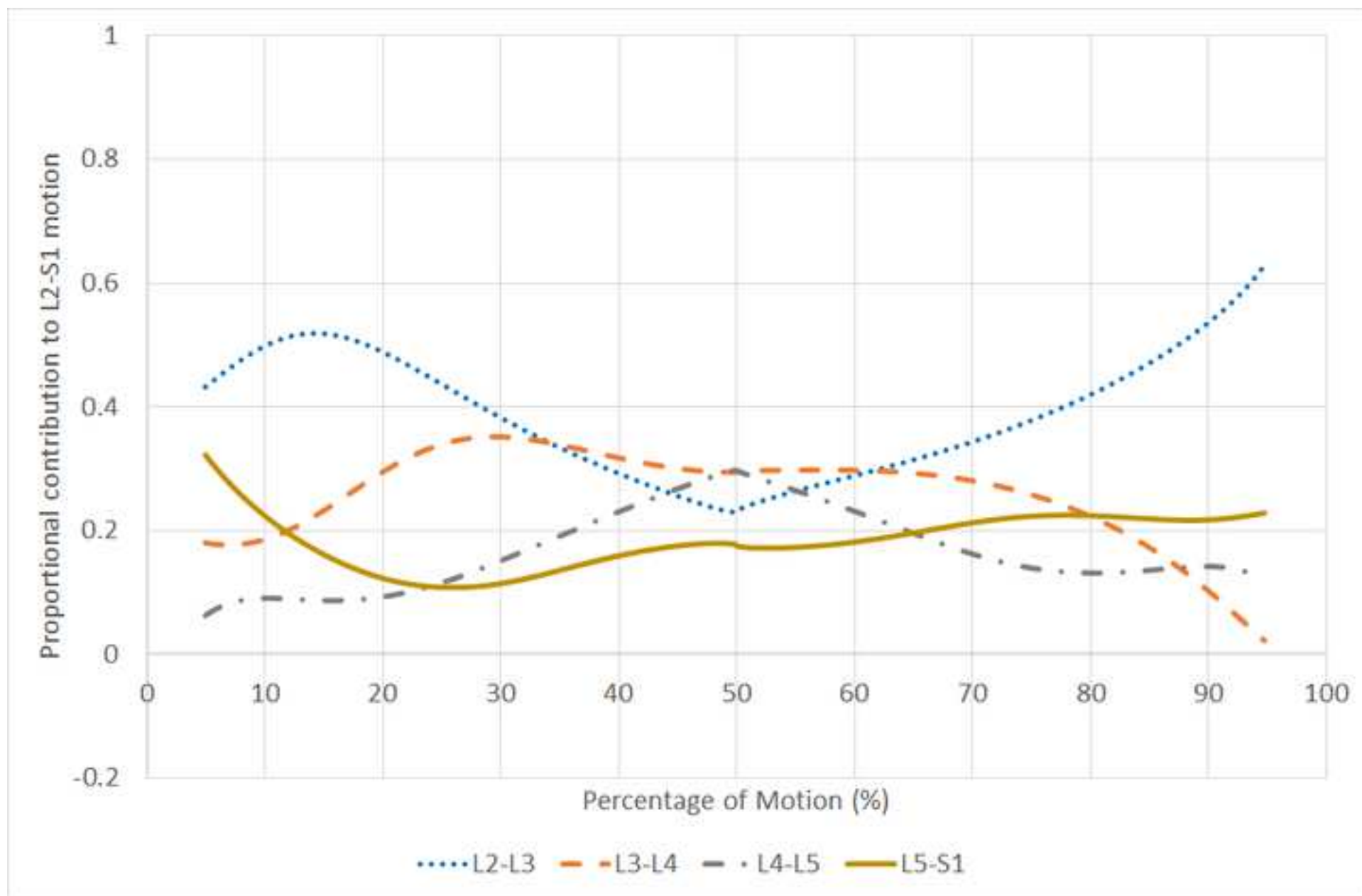
382 (from Breen & Breen 2018).

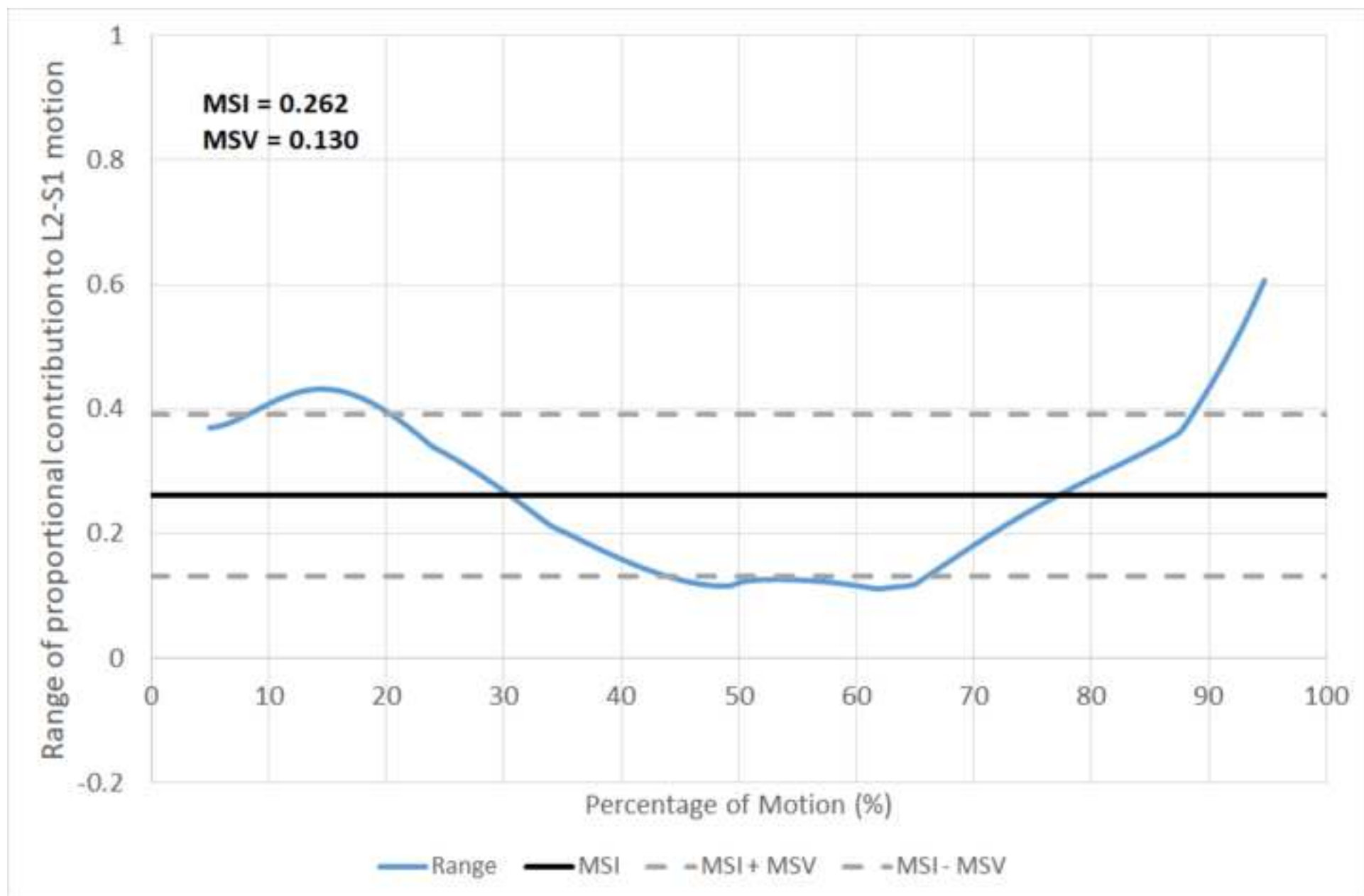
383 Figure 2.

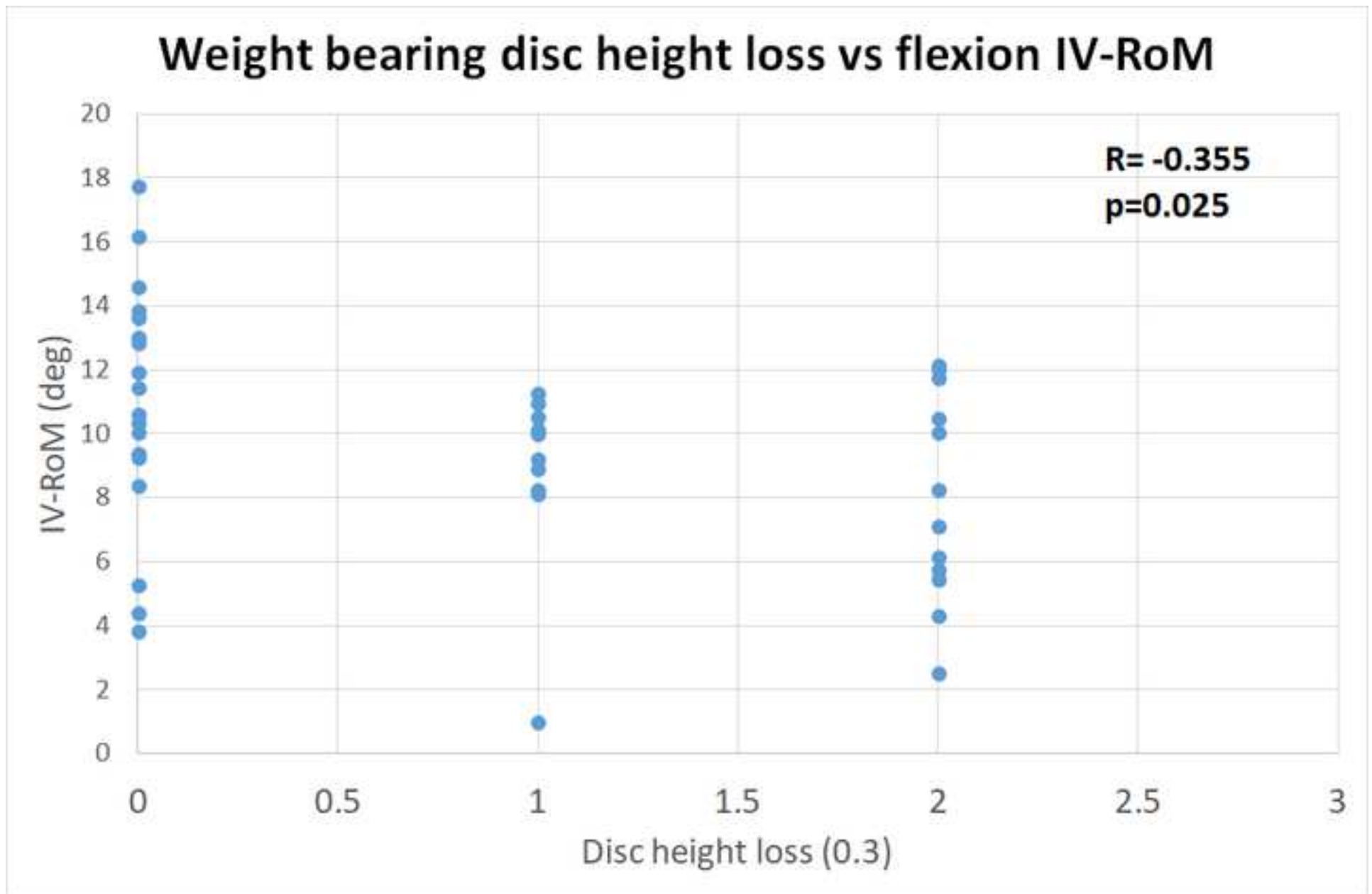
384 Short title: Scatter plots showing correlations between ROM and radiologist weight bearing

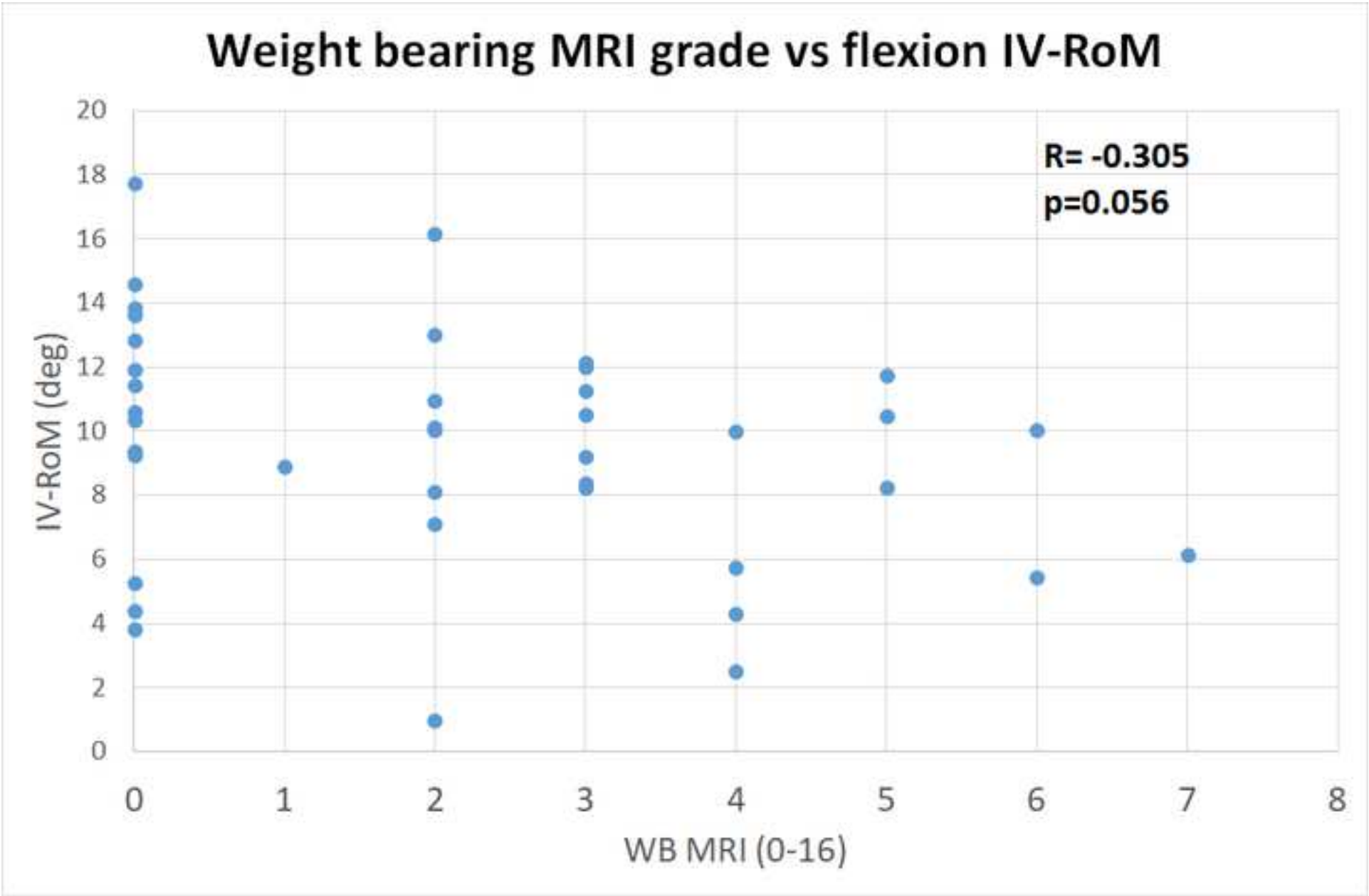
385 MRI assessments of a) disc height loss and b) overall disc degeneration (/16) (Jarosz 1997).











Tables

Table 1

Summary of participant data (n=10)			
	Age	Sex	BMI
	58	M	26.8
	70	M	26.4
	64	F	24.3
	56	F	27.5
	52	F	21.3
	66	F	26.3
	52	F	22.0
	65	F	25.7
	69	F	23.9
	51	F	16.9
Mean	61	2M 8F	24
SD	6.9		3.1
Kinematic scores. significance, median (max) n=40			
	Recumbent	Weight bearing	
Flexion IV-RoM (degrees) p<0.01	4.3 (11.60)	10.0 (17.7)	
Translation (mm) p=0.34	1.89 (3.90)	1.52 (5.34)	
Laxity p=0.435	0.14 (0.37)	0.12 (0.41)	
MSI p=0.20	0.25 (0.56)	0.34 (0.63)	
MSV p=0.01	0.08 (0.15)	0.15 (0.28)	
Composite disc degeneration grade, significance, median (max) n=10			
Radiographic (/16) (NS)	1.9 (4)	2.0 (4)	
MRI (/64) (p=0.04)	5.0 (21)	9.5 (21)	
MRI disc height loss (/12) (p=0.03)	2.5 (7)	3.4 (7)	
MRI T2 signal loss (/12) (NS)	3.6 (7)	3.7 (6)	

Table 2

Weight bearing composite disc degeneration scores				
Scoring system	Mean	Max	Max possible	Max as proportion of upper limit
Composite Kellgren & Lawrence	1.9	4	16	0.25
Composite Jarosz	10.2	21	64	0.33
Jarosz (disc height loss)	3.4	7	12	0.58
Jarosz (T2 signal loss)	3.7	6	12	0.50

Table 3

Correlations* (ρ) between motion sharing MSI/MSV and weight bearing DD				
	Recumbent		Weight bearing	
	MSI	MSV	MSI	MSV
Radiographic	-0.610 (0.06)	0.114 (0.73)	0.165 (0.66)	0.324 (0.37)
MRI DD	-0.241 (0.47)	-0.148 (0.66)	-0.272 (0.43)	0.228 (0.54)
MRI disc heig	-0.317 (0.35)	-0.305 (0.37)	-0.311 (0.37)	0.274 (0.45)
MRI T2 signal	0.310 (0.95)	-0.214 (0.54)	-0.302 (0.37)	-0.076 (0.86)

*Spearman rank correlation