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## Accepted Manuscript

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**Title: Relationships between muscle electrical activity and the control of inter-vertebral motion during a forward bending task**

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ACCEPTED MANUSCRIPT

## Relationships between muscle electrical activity and the control of inter-vertebral motion during a forward bending task

### Abstract

Muscle strengthening exercises are commonly used in primary care for the treatment of chronic, non-specific low back pain (CNSLBP) as it has been theorised that increased muscle activity contributes to the stabilisation of inter-vertebral motion segments during bending and other spinal movements, however this has never been demonstrated *in vivo*.

This study used contemporaneous quantitative fluoroscopy (QF) and surface electromyography (sEMG) to investigate relationships between continuous inter-vertebral motion variables and muscle electrical activity in the lumbar multifidus (LMU), lumbar and thoracic erector spinae (LES and TES) during standardised lumbar flexion and return in 18 healthy male human subjects.

Our results demonstrated that the variability in the sharing of angular motion (i.e. Motion Share Variability MSV) and motion segment laxity during a bending task were significantly ( $p < 0.05$ ) negatively correlated (Spearman) with muscle electrical activity throughout the participant bend for both locally and globally acting muscle groups. MSV was also strongly correlated with L2-3 laxity.

The former suggests a damping mechanism reducing irregular displacements (i.e. less variability in the sharing of segmental motion) during bending and an action of spinal stabilisation by muscles at segmental levels, and the latter a synergy between laxity at L2-3 and MSV. While this has previously been theorised, it has never been shown *in vivo* at the inter-vertebral level. These assessments may be considered for use in validation studies of exercise programs for CNSLBP, however further replication is required.

### Background

Low back pain (LBP) has been linked with spinal instability, and its association with trunk muscle activity has therefore been investigated during numerous tasks. Whilst Ahern et al. (1988) found paraspinal muscle activity to be lower in a low back pain population compared to pain free controls, the consensus is that muscle activity increases in such populations as a stabilisation mechanism (Kuriyama et al. 2005; Sanchez-Zuriaga et al. 2015; Van Dieen et al. 2003). Motor control strategies to stiffen the spine (Gardner-Morse 1995; Cholewicki and McGill 1996) therefore include increasing trunk muscle co-contraction (Granata and Marras 2000), and augmenting local or global paraspinal muscle activation (Bergmark 1989; Reeves

et al. 2006). This provides a rationale for the use of motor control exercises as an intervention in LBP groups (Hodges et al. 1996; Saragiotto et al. 2016).

Whilst the literature supports the idea of training muscular capacity to improve spinal stability, benefits are broadly attributed to the lumbar spine as whole, and there is only limited understanding of the influence of muscle activity on kinematics at segmental levels. Kaigle et al. (1998), using sEMG and spinous pins, studied concurrent lumbar inter-vertebral flexion and return motion and spinal muscle electrical activity in live subjects and found inter-vertebral ranges of motion (IV-RoM) to be reduced with increased muscle activity. Our own group replicated this finding using sEMG and quantitative fluoroscopy (QF). QF is “an objective assessment of the spine in motion using fluoroscopy (moving video x-rays) and automated computer processing algorithms which calculate intersegmental kinematic parameters throughout the motion” (Breen et al. 2012). Utilising QF and sEMG concurrently relationships were found between the timing of the activity of three different spinal muscles and maximum IV-RoM at different segmental levels (Du Rose et al. 2016). QF has also been used to measure the initial rate of the attainment of inter-vertebral rotational motion, referred to in this paper as ‘laxity’, and a parameter termed Motion Sharing Variability (MSV). Laxity is believed by some to represent the dynamic neutral zone (Breen et al. 2015), and MSV is a measure of the variability in how inter-segmental angular rotation is shared across the measured spine throughout a bending cycle (Breen and Breen 2018).

There is evidence from modelling studies that impaired neuromuscular control can leave the lumbar spine vulnerable to buckling under even light loads (Garner-Morse et al. 1995, Cholewicki and McGill 1996). Attention is therefore turning to the relationships between muscle activity and inter-vertebral stability in chronic, non-specific low back pain (CNSLBP). The need is to identify a sub-group that might ultimately benefit from back exercises on the basis of improved inter-vertebral stability. However, IV-RoM is a highly variable parameter and has been found not to discriminate patients with chronic, non-specific low back pain from healthy controls (Mellor et al. 2014). By contrast, the inter-vertebral mid-range measures of inter-vertebral laxity and MSV can be regarded as indicators of reduced restraint and control respectively. While the former is regarded as an expression of motion segment sub-failure (Panjabi 1992), the latter has been shown to be greater in an undifferentiated CNSLBP population than in healthy controls during recumbent bending (Mellor et al. 2014). Laxity can be measured using QF as the initial attainment rate (Teyhen et al. 2005, Mellor et al. 2009, du Rose and Breen 2016) and MSV from multilevel continuous QF studies (Mellor et al. 2014, Breen and Breen 2018). The sEMG data from the back muscles can be recorded contemporaneously.

It can be hypothesised that muscle activity has a damping effect on both laxity and MSV (Reeves et al. 2011) and will be negatively associated with them. Due to the nature of QF imaging, and the requirement to record sEMG concurrently, it was only feasible to measure these parameters during a single plane of motion, so that ionising radiation dose received

by any one participant was minimal. Forward bending is the most commonly evaluated task when investigating lumbar biomechanics, and was therefore considered the most appropriate movement for study. The aim of this investigation was therefore to use QF and sEMG concurrently, to determine whether relationships exist between kinematic motion parameters (i.e. MSV and laxity) and mean paraspinal muscle activity recorded during a standardised forward bending task.

## Methods

Twenty males with no recent history of low back pain were recruited from the AECC University College student population. Ethical approval was provided by the National Research Ethics Service (Bristol 10/H0106/65), and all participants gave written consent. The inclusion and exclusion criteria are outlined in Table 1.

Table 1. Eligibility Criteria

### Data collection

Quantitative fluoroscopy and surface electromyography were used concurrently to acquire lumbar inter-vertebral images and record paraspinal myoelectric activity.

### Surface electromyography (sEMG)

Prior to the image acquisition, participants' skin was prepared for the application of sEMG electrodes by light abrasion, cleaning with alcohol and when necessary shaving of the area. Disposable Ag-AgCl electrodes were then bilaterally applied using a 20mm centre to centre inter-electrode distance, to the thoracic erector spinae (TES) (5cm lateral to the T9 spinous process), the lumbar erector spinae (LES) (2cm lateral to the L2 spinous process), and the superficial lumbar multifidus (LMU) (2cm lateral to L5 spinous process, along a line between the posterior superior iliac spine and the spinous process of L1). Biopac wireless transmitters (Bionomadix Dual Channel Wireless EMG) were fastened to the lower back with the use of Velcro adhesive pads. As there was found to be no significant difference between left and right sides at any level during the bending task, the average of the mean amplitudes recorded from both sides was used in the analysis.

The sEMG signals were recorded using a sampling rate of 2000 Hz, a common mode rejection ratio (CMRR) of 110 dB and an input impedance of 1000MΩ. All sEMG signals were band pass filtered (10-500Hz) and full wave rectified. Smoothing was applied with a time constant of 300 ms, and the mean root mean square (RMS) amplitude was then calculated over the twenty second duration of each bending cycle, normalised to a sub-maximal voluntary contraction (sMVC), and expressed as a percentage of this contraction. To obtain the sMVC, each participant was asked to lie prone with their hands behind their

head. They then raised their torso off the bench and held for five seconds whilst their legs and pelvis were stabilised. The procedure was repeated three times, and the average recording was taken as the reference contraction value (sMVC).

#### Image acquisition and processing

A Siemens Arcadis Avantic VC10A digital fluoroscope (CE0123) was used to collect the fluoroscopic images at 15Hz during a standardised sagittal forward bending and return task. Participants were guided at a constant rate through a range of 60° of flexion, and the return to an upright neutral position, by following a rotating motion frame (Figure 1). Myoelectric paraspinal activity was recorded concomitantly. The QF motion frame and the sEMG recordings were synchronised with the use of a bespoke trip switch attached to the motion frame. When the motion frame began to move, a data point was registered on the sEMG timeline. The entire bending sequence was approximately 20 seconds in duration.

Figure 1: Motion frame apparatus.

Participants were asked to stand with their right hand side next to a motion frame, and to place their forearms on a rotating arm rest. Practice flexion and return sequences (without radiation) were then performed at 20° increments to ensure participant tolerance. Upon commencement of image recording, the motion frame guided each participant through 60° of forward flexion and the return to a neutral upright position. The pelvis was restrained using a belt applied to the anterior superior iliac spine (ASIS) attached to the motion frame, and a bracing pad applied to the lower sacral segments. The image field was positioned such that all motion segments between L2 and S1 were visible in the image field throughout the bending sequence. A lead apron was worn to protect the gonad region.

Image sequences were then transferred to a desktop computer and analysed using bespoke coding written in Matlab (The Mathworks, Cambridge), during which templates were manually created around each vertebral outline from the first image, a process repeated five times to increase precision. Subsequent image frames could then be tracked automatically, producing a continual recording of the template movement throughout the bending sequence. A simple output from this is angular displacement is angular displacement at the inter-vertebral level over time (Figure 2). For analysis, the data extracted comprised the laxity over the first 10° of inter-vertebral motion for levels L2-3, L3-4, L4-5 and L5-S1 (Figure 3), and the MSV.

Figure 2: A typical example of angular displacement at the inter-vertebral level over time (represented by image number).

Note: While this figure demonstrates a typical example of angular displacement at the inter-vertebral level over time. Each participant demonstrates unique motion characteristics

including rate/range of motion and start time of individual joints. Change in rotation in the flexion direction is considered negative as the angle between two adjacent vertebrae typically decreases during forward flexion.

Figure 3: Laxity calculation: A typical example of laxity, calculated as the gradient of the linear trend of inter-vertebral motion as a function of motion frame angle for the first 10 degrees over which the intervertebral level is in motion.

Motion Sharing is calculated as the relative contribution to motion of a single level as a function of the whole measured spine at each point in time (e.g. L2-L3 angular displacement at time point  $t \div$  Sum of all L2-S1 angular displacement at time point  $t$ ).

Because segmental angular displacements are small at the beginning of participants bend and as they return to their original position, and are close to the precision limit of the QF systems (0.52 degrees, Breen 2006), contribution to motion at these time points are truncated to remove large relative contribution to motion errors. The range of contributions to motion is found at each time point throughout the motion after filtering to remove errors (Figure 4), MSV is calculated as the square root of the variance of filtered range of contributions across all data points in each sequence (Breen and Breen 2018).

Figure 4: Filtered Range of Contributions (fRC) displayed against image frame number.

Note: Motion sharing contributions to angular displacement for each intervertebral level over time (image frame number, 15 frames per second). To remove error amplification at the initial and final parts of the sequence, proportional values are filtered to include only the middle 80th percentile of the rate of change of the proportional contribution of an individual intervertebral joint angle to the sum of the intervertebral angles between L2 and S1. This is calculated as the first derivative of a level's proportional contribution to position in an image frame. Further details of this process are outlined elsewhere (Breen and Breen 2018). The range between the contributions to motion sharing is calculated after filtering.

## Results

The sEMG recordings and fluoroscopic images of 18 males, mean age of 27.6 years SD (4.4), mean height of 1.8 m SD (0.06), and mean BMI of 24 SD (2.2), with no history of low back pain were included in the data analysis. The mean radiographic exposure factors for the cohort were documented as 79.7 kV SD (5.4) and 55.4 mA SD (3.4), and the mean effective dose was 0.143 mSv, calculated using ICRP103 conversion software PCXMC (Monte Carlo Simulation Package). Data sets were not included for two participants. This was due to the need for continuous motion capture of each intersegmental level throughout the motion sequence. For these two participants, at least one vertebra was not identifiable by the tracking programs for more than 1 frame during the bend. MSV and laxity were tested for



normality using the Shapiro Wilk test. Since no evidence of normality could be statistically proven for the data sets, Spearman rank correlations were used.

#### Correlations between MSV and muscle activity

The correlations between MSV and muscle activity at all recording sites are shown in table 2. Moderate negative relationships with MSV were found with all muscles ( $r$  values ranging between -0.431 and -0.659), however statistical significance was not reached with the superficial lumbar multifidus. Statistically significant relationships were found however with the thoracic and lumbar erector spinae (Table 2).

Table 2: Correlations between MSV and muscle activity (Spearman rank)

#### Correlations between muscle activity and laxity

Statistically significant negative correlations between muscle activity and laxity were found. These moderate negative relationships ( $r$  values ranging between -0.588 and -.75), were consistent for all muscles with laxity at the level of L2-L3. Significance was also reached for the thoracic erector spinae and laxity at the level of L3-L4 (Table 3). Relationships approaching significance were also shown between lumbar erector spinae and laxity at L3-L4 ( $p = 0.051$ ) and between the superficial lumbar multifidus and laxity at the level of L5-S1 ( $p = 0.055$ ). The strongest correlation found was between MSV and laxity at the level of L2-L3, with an  $r$  value approaching 0.8.

Table 3: Correlations between MSV, muscle activity and laxity (Spearman rank)

All significant correlations were further analysed using simple linear regression, and the effects of mean muscle activity on both MSV and laxity were calculated. This yielded  $r^2$  values ranging between 0.208 and 0.402, as shown in scatter plots representing all significant relationships Figure 5 (A-G).

Figure 5: Scatter plots showing relationships between muscle activity, MSV and laxity parameters

## Discussion

By using QF and sEMG concurrently it was possible to investigate relationships between active and passive sub-systems at an inter-vertebral level, providing a unique insight into possible spinal stabilisation mechanisms in a non-low back pain group. The analysis showed statistically significant negative correlations between both muscle activity and laxity, and between muscle activity and MSV. As a surrogate for the neutral zone, laxity represents

motion segment attainment rate during the initial stages of inter-vertebral rotation, and therefore the decrease in muscle activity associated with an increase in this variable supports the hypothesis that reduced muscle activity is associated with both reduced control and restraint. Previous studies have shown an apparent co-dependence amongst motion segments in terms of both angular ranges of motion and laxity parameters, showing for example how when laxity increases at L2-L3, there is a subsequent decrease in laxity at L4-L5 and vice versa (Du Rose and Breen 2016). Considering this apparent inter-dependency, it is possible that when segments (such as L2-L3) show increased laxity, that motion segments elsewhere will have to adapt their behaviour in accordance. This could feasibly partly explain the increase in motion share variability associated with increased laxity at this level. The fact that a decrease in muscle activity also correlates with an increase in MSV, would suggest that increased muscle activity is a mechanism employed to achieve spinal stability by controlling such variability.

This is in agreement with previous spinal stabilisation theories, but this is the first time that such mechanisms have been demonstrated *in vivo* at the inter-vertebral level. Garner-Morse (1995) for example suggested that activated muscles act like stabilising springs that reduce the requirement for active neuromuscular responses to small changes in the system (Gardner-Morse et al. 1995). The relationship between muscle activity and spinal stiffness is also well documented elsewhere in the literature (Ross et al. 2015; Gardner-Morse et al. 1995; Stokes and Gardner-Morse 2001), and has been related to the feedback provided by spinal positioning, a static interpretation of stability (Reeves et al. 2011). Reeves and Cholewicki (2010) however, also consider the concept of 'damping' (i.e. a mechanism of spinal control related to velocity feedback), a phenomenon they suggest should be more regularly considered, especially in terms of more dynamic spinal stability investigations (Reeves and Cholewicki 2010).

The results of this study suggest that such a damping mechanism may influence lumbar kinematics during a forward bending task, and may help explain the relationship found between MSV and paraspinal activity. As increased variability intuitively relates to the degree of muscle spindle feedback (Nitz and Peck 1986; Buxton and Peck 1989), it is likely that beyond certain thresholds, an increased motor activity response is required to stabilise the spinal system, which may have knock on consequences in terms of tissue loading (Granata and Marras 2000). Regardless of the mechanism, faced with damage to any component of this feedback mechanism (e.g. muscle spindles), such a stabilisation mechanism would be compromised, which could feasibly result in an increased MSV in such low back pain populations.

Interestingly, weak to moderately strong relationships between MSV and paraspinal activity were found in both locally acting (LMU and LES) and globally acting (TES) muscles. This is in contrast to the distinct stabilisation roles attributed to local and global musculature proposed by Bergmark (1989). Indeed, Van Dieen et al. (2003) proposed that patients with

LBP increase spinal stiffness by increasing superficial muscle activity (Van Dieen et al. 2003), whilst there are those that have argued that rehabilitation of the deeper muscles is important in terms of optimising spinal control (Richardson and Jull 1995). The findings of this study however would suggest that all extensor musculature (globally and locally acting) have a controlling influence, as all recorded muscle activity was negatively correlated with MSV, providing a possible explanation as to why, despite wide variation in study methodologies (e.g. different electrode attachment sites representing the same muscle), the same generic conclusions are reached (i.e. that increased muscle activity is a strategy for spinal stabilisation).

The findings also offer a reason why the majority of exercise interventions will typically result in improved patient outcomes. As increased MSV has been shown to be associated with CNSLBP (Mellor et al. 2014), exercise programmes that influence any of the paraspinal muscles studied, may also reduce motion share variability and possibly reduce the symptoms associated with the LBP. Indeed, whilst it is accepted that exercise interventions are beneficial to low back pain populations (Gordon and Bloxham 2016), there remains much debate over which type of exercise programme is superior (Saragiotto et al. 2016; Costa et al. 2009; Bronfort et al. 2011; Macedo et al. 2012). Further investigations are required to determine if CNSLBP patients with high MSV levels represent a subgroup that respond to such programs.

### **Limitations**

The study's results are limited as they show measurements taken from a relatively small, low back pain free, young adult male population. Consideration must also be given to the fact that the correlations found may not indicate causal relationships, and therefore any extrapolation of the results into low back pain populations are purely theoretical, and should only be considered as such. In addition, the study only considered relationships during sagittal forward bending, and so provided no insight into relationships in other planes of movement or other functional tasks.

Restricting the study to a male only population was primarily to reduce the impact of the greater variability in Soft Tissue Thickness (STT) associated with females, but also as an all-male team, the use of female participants would have necessitated another 'female' member of staff to be in attendance for chaperone reasons. This was not possible for due to both resource and space restrictions. We would like to note however that future planned studies will involve larger cohorts, and include both genders. The use of sEMG is also limited in terms of distinguishing between deep and superficial musculature. In this study, sEMG recorded at the level of L5 was taken to represent the activity of the lumbar multifidus. The recording however most likely represents only the most superficial multifidus, and as the deep and superficial multifidus are purported to have different spinal stabilisation roles (Moseley et al. 2002), comments about deep muscle activity should be taken in context.

## Conclusion

Using QF to determine novel kinematic parameters (utilising inter-vertebral information), combined with sEMG, it was possible to determine relationships between intersegmental laxity, MSV and paraspinal muscle activation. This supports previous work that has suggested increased paraspinal muscle activation to be a mechanism of spinal stabilisation; however, this is the first time this has been demonstrated at an inter-vertebral level *in vivo*. The potential links between parameters such as laxity, MSV and CNSLBP would suggest that these assessments might be considered for use in validation studies of exercise programs for CNSLBP. However, this is the first study to demonstrate this and replication is suggested using forward bending and other functional tasks before embarkation on such validation studies can be recommended.

## Conflict of interest

The authors declare that there is no conflict of interest.

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Autobiography: Alister du Rose

Alister du Rose is a senior lecturer at the University of South Wales (USW) within the Faculty of Life Sciences and Education, where he teaches on Research and Clinical Management modules.

Prior to joining the USW, Alister completed his PhD studies at the University of Bournemouth in the area of spinal biomechanics, using fluoroscopy and surface electromyography concurrently to look for relationships between spinal kinematics and paraspinal myoelectrical activity.

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Figure 1: Standardised motion frame



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Figure 2: A typical example of angular displacement at the inter-vertebral level over time (represented by image number).

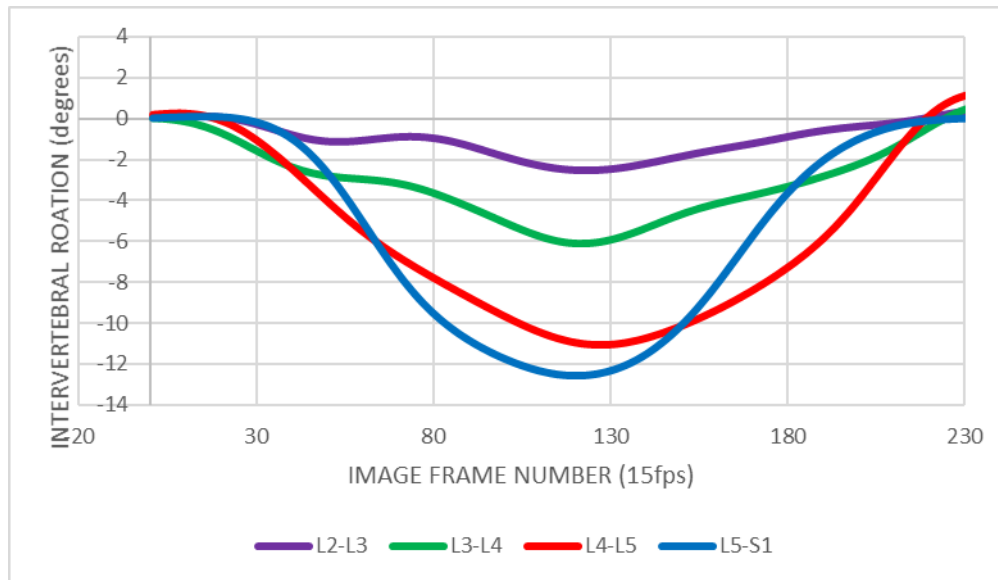


Figure 3: Laxity calculation: A typical example of laxity, calculated as the gradient of the linear trend of inter-vertebral motion as a function of motion frame angle for the first 10 degrees over which the intervertebral level is in motion.

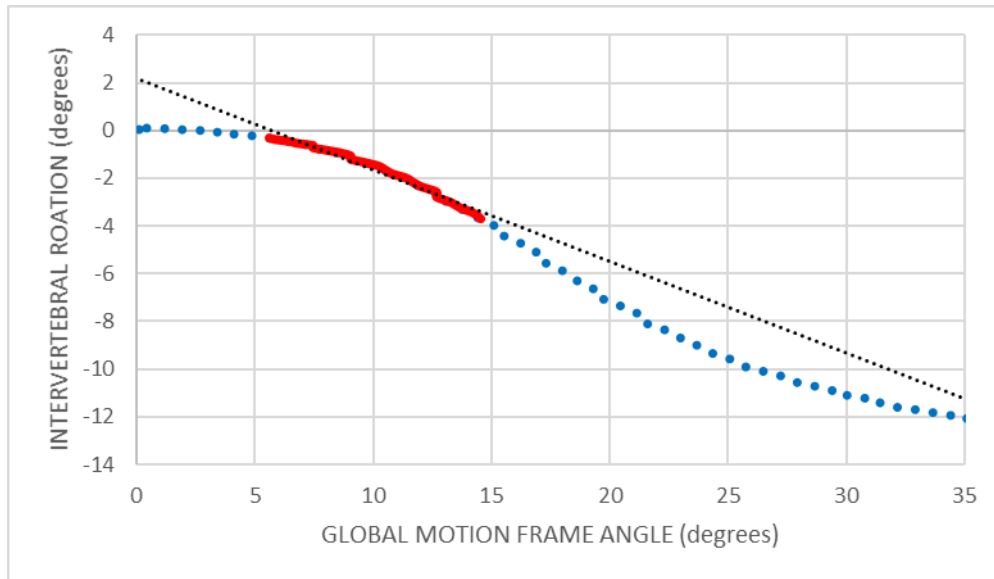


Figure 4: Filtered Range of Contributions (fRC) displayed against image frame number.

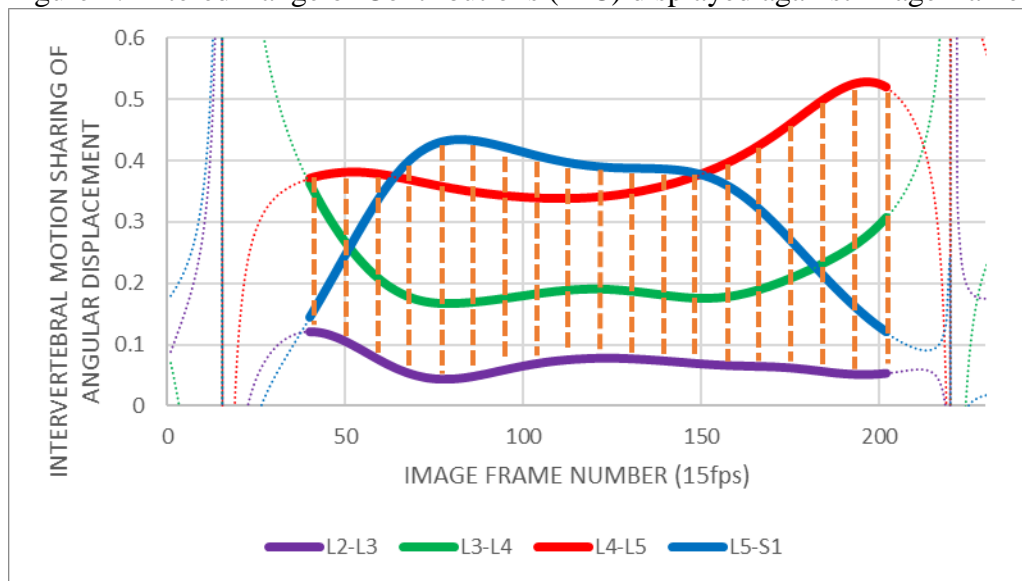


Figure 5: Scatter plots showing relationships between muscle activity, MSV and laxity parameters

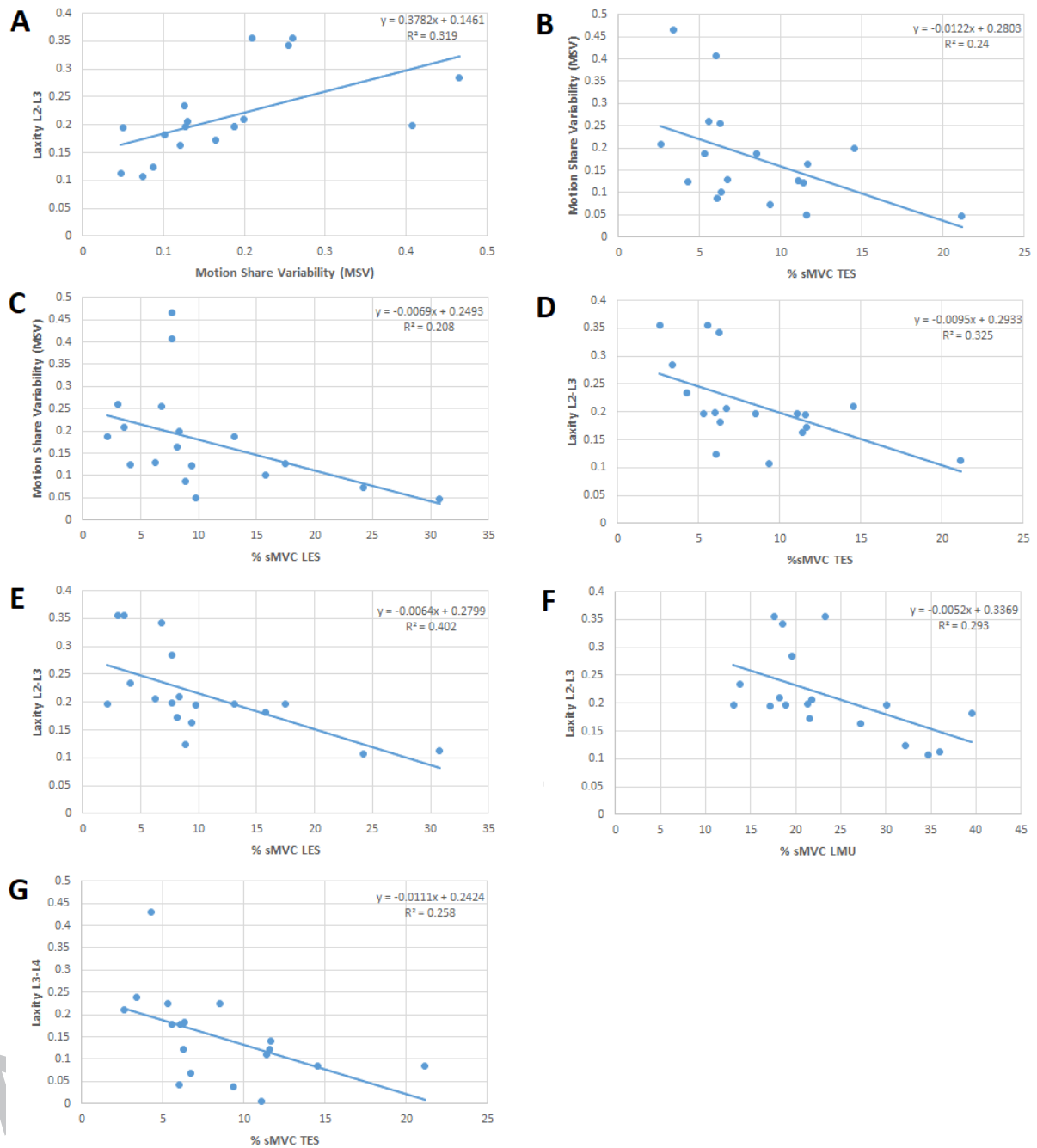


Table 1: Eligibility Criteria

Inclusion	Exclusion
Male (aged 20-40 years)	Poor understanding of English
Ability to understand written English	Ongoing treatment for osteoporosis
Willing to participate and capable of providing informed consent	History of spinal, abdominal or pelvic surgery
BMI less than 30	BMI greater than 30
No history of low back pain (that affected ADL's for at least one day over previous year)	Exposure to medical radiation greater than 8mSv within the past 2 years

Table 2: Correlations between MSV and muscle activity (Spearman rank)

		% sMVC TES	% sMVC LES	% sMVC LMU
MSV	r	<b>-0.543</b>	<b>-0.659</b>	-0.431
	p	<b>0.02</b>	<b>0.003</b>	0.074

Table 3: Correlations between MSV, muscle activity and laxity (Spearman rank)

		Laxity L2-L3	Laxity L3-L4	Laxity L4-L5	Laxity L5-S1
MSV	r	<b>0.798</b>	0.236	-0.225	0.105
	p	<b>0.000</b>	0.347	0.369	0.677
% sMVC TES	r	<b>-0.617</b>	<b>-0.611</b>	-0.06	-0.095
	p	<b>0.006</b>	<b>0.007</b>	0.813	0.708
% sMVC LES	r	<b>-0.75</b>	-0.467	-0.008	-0.085
	p	<b>0.000</b>	0.051	0.974	0.738
% sMVC LMU	r	<b>-0.588</b>	-0.441	-0.026	-0.459

p

**0.01**

0.067

0.919

0.055

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