

## VISCOELASTIC RESPONSES OF THE LUMBAR SPINE DURING PROLONGED STOOPING

Gwanseob Shin<sup>a</sup>, Gary A. Mirka<sup>a</sup>, and Elizabeth G. Lobo<sup>a,b</sup>

<sup>a</sup>The Ergonomics Laboratory, Industrial Engineering Department, North Carolina State University

<sup>b</sup>Joint Department of Biomedical Engineering, North Carolina State University and UNC Chapel Hill  
Raleigh, North Carolina

There is considerable evidence that awkward postures of the low back are related to the incidence of low back disorders (LBDs), but the specific biomechanics/physiology of this link is not fully developed. This study combined empirical work with finite element analyses to explore this relationship. The empirical work focused on quantifying the time-dependent responses of the lumbar spine during a prolonged stooped posture by assessing the changes in the sagittal plane range of motion and the electromyographic activity of the back extensor musculature during and after prolonged stooping. Ten healthy participants performed a regimen of a 10 minute stooping period followed by a 10 minute upright standing recovery period, with an isokinetic lift at every 2.5 minutes. Results showed significant creep effects of the flexion angle and the increased activity of extensor muscles in stooping to compensate for the reduced extensor moment producing capability of the passive tissues. The 10 minute upright standing did not produce a full recovery of the lumbar spine tissues but a 30 second rest break in the middle of the stooping moderated these viscoelastic responses. A nonlinear viscoelastic 3D finite element (FE) model of the lumbar spine was developed to predict the responses of the passive and active tissues of the low back. Validation of the FE model by comparing its predicted results (range of motion, muscle activation levels, etc.) with experimental results indicated good agreement in terms of mechanical behaviors in stooping, confirming the capability of the FE model as a potential tool for risk assessment of the prolonged stooping tasks.

### INTRODUCTION

The stooped (i.e. fully flexed) posture of the lumbar spine has been shown to impact the biomechanics and physiology of the tissues of the low back and it is hypothesized that these changes can contribute to the development of low back pain (LBP) and disorders (LBDs) in workers in occupations such as construction and agriculture. In the stooped posture, the upper body weight is supported by the passively generated extension moment from the spinal ligaments and muscle-tendon units in the posterior side of the spine (Kippers and Parker, 1984; McGill and Kippers, 1994). Since most spinal tissues are viscoelastic, prolonged stooping can initiate creep deformation (in constant stress) or stress-relaxation (in constant strain) of the passive tissues (Adams and Dolan, 1996). The passive stretch can cause micro tissue damage and acute inflammation in the spinal ligaments. These mechanical and physiological changes in the passive tissues can impair lumbar spine stability and require greater activation of muscles to maintain the spinal stability and protect the damaged tissues (Solomonow, 2004). However, the passively stretched extensor muscles in stooped posture may lose their force-generating capacity (Fowles, Sale, & MacDougall, 2000) and this can lead to reduced strength performance of back extensor muscles. Greater demand on weaker muscles can result in faster fatigue generation or failure to sustain lumbar stability, specifically when the lumbar spine is subjected to high external load right after the prolonged stooping.

Identifying variables impacting the relationship between the prolonged stooping posture (e.g. duration, rest-breaks, extension moment, etc.) and the viscoelastic mechanical response (e.g. creep response, muscle activation profiles, etc.)

is an important step to understand the injury mechanism and assess risks of the prolonged stooping for low back disorders. The lumbar spine is a complicated structure consisting of many soft tissues of which material properties are nonlinear and viscoelastic. Due to the complexity of the structure and difficulties to incorporate many internal/external variables, computational modeling and simulation using a finite element (FE) model can be a proper method that can quantify responses of the lumbar spine in the prolonged stooping. The objective of this study was two-fold. First, this study examined time-dependent changes of the full lumbar flexion angle and extensor muscle activities during prolonged stooping and recovery standing to understand viscoelastic responses in the in-vivo human lumbar spine. Second, a 3D finite element (FE) model of the lumbar spine was developed and validated as a predictive tool to assess physical risks of the prolonged stooping.

### METHODS

#### Participants

Five male and five female participants (mean age: 27.8 years, SD of age: 2.7) were recruited from the university community. All participants gave written informed consent prior to participation in the experiment. Potential participants were excluded from consideration if they had any chronic or acute back pain. Also, because the physical properties of the passive tissues can vary with age, participants under 20 and over 35 years old age were not be recruited to minimize variation due to age.

## Apparatus

The electromyographic activity (EMG) of lumbar extensors were collected using four pairs of surface electrodes which were placed on skin at 5 cm lateral from the midline of vertebrae at L3 level for erector spinae and 3 cm lateral from the midline at L4 level for multifidus on both right and left side. The lumbar flexion angle was measured by an electromagnetic motion tracking system (MotionStar, Ascension Technologies). Relative angle between two magnetic sensors that were located on the skin surface overlying the spinous processes of T12/L1 and L5/S1 was defined as the lumbar flexion angle.

## Procedures

This experiment consisted of two days of data collection: one day without a rest break (no-rest condition) and the other day with a rest break (w/rest condition). The order of the no-rest and w/rest day was randomly assigned to participants. These data collection days were separated by one-week to allow complete recovery of passive tissues.

Participants began the study by performing maximum voluntary contraction exertions in an isokinetic dynamometer. EMG data collected during these exertions were used in the normalization process. During the experiments, participants were secured to a fixture that was designed to limit pelvic rotation (Figure 1). Data collection consisted of two phases; a 10 minute prolonged stooping (static full flexion) phase and a 10 minute upright standing recovery phase. Before the stooping phase began, participants performed a baseline full flexion motion and then performed a baseline isokinetic lift (time '0'). During these exertions, the participants flexed their back forward slowly and reached the full flexion posture. When the full flexion posture was made, participants were asked to put their heads down, relax, and exhale (McGill & Brown, 1992), and the lumbar flexion angle was measured for 5 seconds (Figure 1). Next, a weight box was handed to participants and they lifted the box from the full flexion posture to 30° trunk flexion angle in a period of 15 second with consistent lifting speed. The 15 second duration for the lift was set to simulate continuous quasi-isometric weight holding postures and minimize the motion artifacts of EMG. The weight of the box was set at 40% of their posture specific capacity. The box was taken by the experimenter when the lift was finished.

The prolonged stooping phase started as soon as participants finished the isokinetic lift and returned to the full flexion posture. The stooped posture continued for 10 minutes with the aforementioned isokinetic lift occurring at 2.5 minute intervals. The full lumbar flexion angle was measured before each lift and the EMG and angle were simultaneously collected during the lifting motion. In the day with a rest break, a 30-second upright standing rest break was given at 5<sup>th</sup> minute of stooping phase (after the second isokinetic lift), while no such break was given on the other day. At the end of 4<sup>th</sup> lift (10<sup>th</sup> minute) participants stood up to upright standing posture and the recovery phase began. The recovery phase continued for 10 minutes and participants performed a 10

second static full flexion posture and an isokinetic lift at 2.5 minute intervals during this recovery phase. Participants returned to the upright standing posture after each lift to continue recovery.

## Data Processing

The EMG signal was full wave rectified and filtered using 6 Hz Butterworth second-order low-pass filter to produce a linear envelope (Winter, 2005). The EMG were then normalized to the muscle specific MVC values. The bilateral pairs were then averaged to generate a values for that muscle group. In order to align the normalized EMG data with the FOB angle data, the number of EMG samples was reduced to 42.66 per second by taking average of windows with 24 data points ( $1024/42.66 = 24$ ) width. The muscle activity variables were then calculated as the average of the normalized EMG values that occurred as the subject moved through the 1°-3° with 0° being defined as the full flexion angle during the baseline (i.e. pre-stooping phase) exertion.

## Data analysis

The effect of the independent variables TIME and REST BREAK were tested using a within subject design. The TIME effect was tested within each rest condition after separating data set into no-rest condition and w/rest condition groups. The rest break effect was tested with the data set between 5<sup>th</sup> and 20<sup>th</sup> minute. The assumptions of the ANOVA technique were verified using the graphical techniques advocated by Montgomery (2001).



Figure 1. Full lumbar flexion (stooped) posture.

## FINITE ELEMENT MODEL

### Modeling

A 3D nonlinear viscoelastic FE model of the lumbar spine which consists of five vertebrae (L1 to L5), five intervertebral discs (L1/L2 to L5/S1), posterior ligaments (posterior

longitudinal ligament, capsular ligament, ligament flavum, intertransverse ligament, interspinous ligament, supraspinous ligament), and lumbar extensor muscles (multifidus, erector spinae) was created using ABAQUS v6.4. Geometry and material properties were taken from previous in-vitro and FE model studies (Marchand & Ahmed, 1990; Panjabi, Goel, Oxland, Takata, Duranceau, Krag, & Price, 1992; Bogduk, Macintosh, & Percy, 1992; Shirazi-Adl, Ahmed, & Shrivastava, 1986; Goel, Monroe, Gilbertson, & Brinckmann, 1995; Wang, Parnianpour, Shirazi-Adl, Engin, Li, & Patwardhan, 1997). Since geometry and boundary conditions are symmetric to the mid-sagittal plane, only the right half of the lumbar spine was created and the symmetry boundary conditions were applied on the mid-sagittal plane.

### Loading

The model simulated the tasks of the in-vivo experiments; 30 minutes standing as a preload, 10 minutes stooping, 10 minutes recovery standing, and a weight holding task at every 2.5 minutes during the stooping and recovery.

The full lumbar flexion (stooped) posture was acquired when a flexion moment by an upper body weight (400 N) on the center of gravity of torso-arms is balanced by an extension moment by passive resistance of the spine, which changed over the duration of the stooping due to the viscoelastic properties of spinal tissues. A full lumbar flexion angle at the beginning of stooping was defined as an initial lumbar flexion angle.

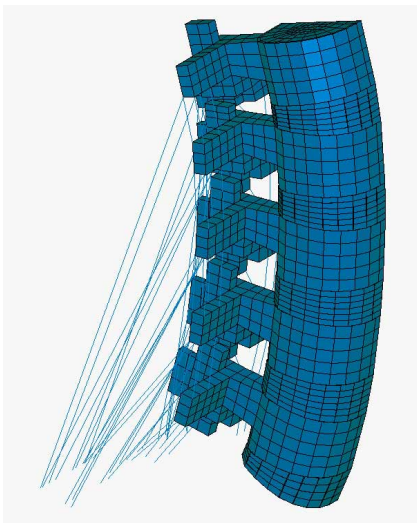


Figure 2. FE model of the lumbar spine.

At every 2.5 minutes from the beginning of the stooping, the lumbar spine was extended  $2^\circ$  over the initial full flexion angle (time '0') and the origin nodes of back extensor muscles were fixed in all directions to simulate active forces of lumbar extensors at weight holding. Then, an additional 250 N vertical force was added to the center of gravity of torso-arms as an external weight on hands so the flexion moment by an upper body weight and the additional load was supported by passive resistance of the spine and the reaction forces on the

muscle origin nodes. The bottom surface of L5/S1 disc and the end nodes of ligament and muscle elements were fixed in all directions throughout the simulations.

## RESULTS

### Full lumbar flexion angle (in-vivo)

Mean full lumbar flexion angle during stooping and recovery was significantly different ( $p < 0.05$ ) between TIME levels in both rest conditions. Figure 3 shows the creep of the full lumbar flexion angle in stooping and recovery. The effect of the rest break was also significant. Mean full flexion angle between 7.5<sup>th</sup> minute and 15<sup>th</sup> minute was significantly less in w/rest condition than the angle in no-rest condition.

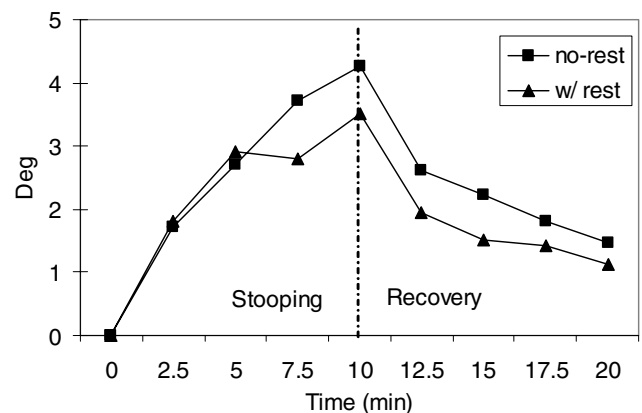


Figure 3. Creep of the full lumbar flexion angle.

### EMG of extensor muscles

The normalized EMG measures increased during stooping and decreased toward the initial level during recovery (Figures 4 and 5). Influences of the time duration of stooping and recovery on the normalized EMG of multifidus and erector spinae muscles were significant in both rest conditions. The rest break effect was statistically significant only at 12.5<sup>th</sup> minute and between 10 and 12.5<sup>th</sup> minute for multifidus and erector spinae, respectively.

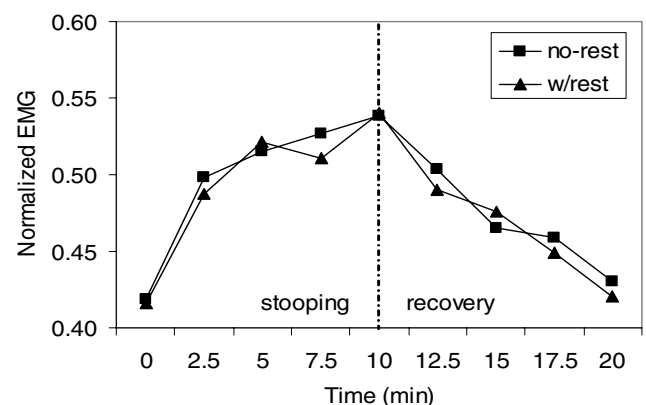


Figure 4. Normalized EMG of multifidus.

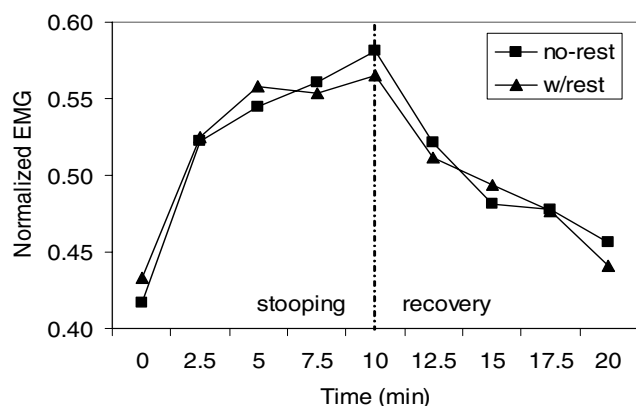


Figure 5. Normalized EMG of erector spinae.

### Predicted full lumbar flexion angle by FE model

Full lumbar flexion angle was defined by the relative angle of L1 top surface to upright standing posture in sagittal plane. The FE model predicted the increase of the angle during stooping and decrease during recovery, similar to observed experimental data. Figure 6 and 7 compare the predicted full flexion angle by the FE model with in-vivo observations.

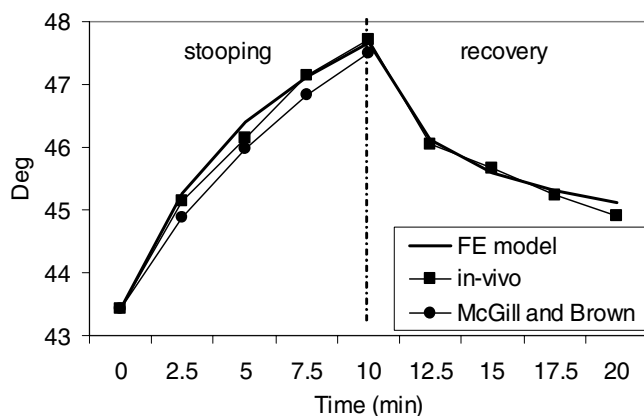


Figure 6. Predicted full flexion (no-rest condition).

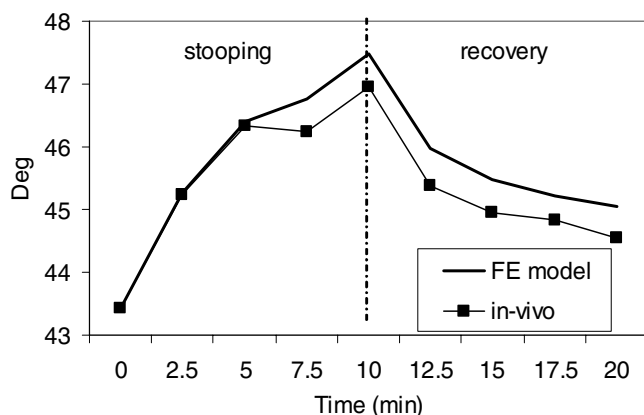


Figure 7. Predicted full flexion (w/rest condition).

### Predicted muscle forces by FE model

Square root of sum of squares (SRSS) of x, y, and z directional reaction forces were obtained from all muscle origin nodes at each weight holding step, and the SRSS reaction forces were averaged within each muscle group. The predicted force data were compared with in-vivo EMG data of this study (Figures 8 and 9). Because of the different orientation of force vectors between the SRSS reaction force and the muscle contraction force in experimental study, the two data sets were normalized relative to their initial values at time 0, and the ratios of the two were compared. Predicted reaction forces of all muscle groups were averaged because no apparent difference in the reaction force between muscles groups was observed.

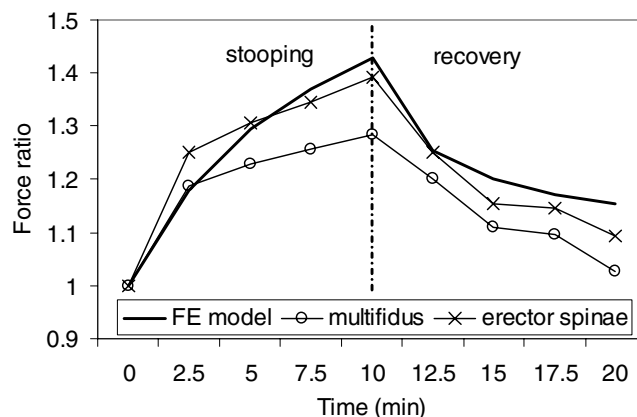


Figure 8. Ratio of predicted force with in-vivo EMG (no-rest condition).

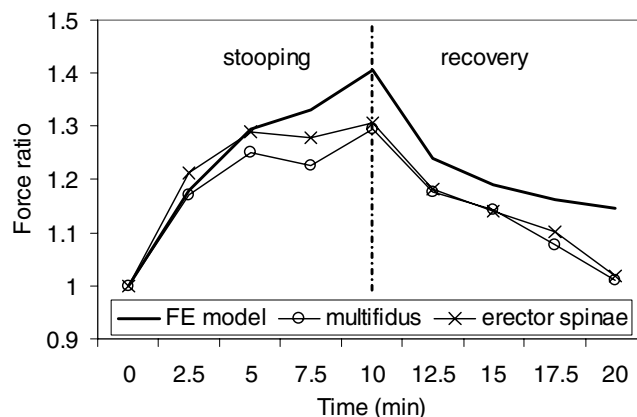


Figure 9. Ratio of predicted force with in-vivo EMG (w/rest condition).

### DISCUSSION

The increase of the full lumbar flexion angle during stooping indicated creep deformation of passive tissues and this resulted in a reduced contribution of the passive tissues to generating extension moment at a given (non-maximal) trunk flexion angle. Transferring extension moment from the

passive tissues to active muscles was observed by the analysis of EMG of trunk extensors. Changes of normalized EMG in stooping and recovery resembled the responses of the full lumbar flexion angle. In the 10 minute stooping, the normalized EMG of multifidus and erector spinae muscles increased 28.6 % and 39.3 %, respectively, suggesting that muscles were required to generate more active forces to compensate for the loss of contribution of passive tissues (Adams and Dolan, 1996). Larger full lumbar flexion angle indicated more slack in the lumbar spine after stooping, and higher muscle activity level was a sign of reduced contribution of passive tissues in generating extension moment. It is believed that lumbar spine instability could be created by these mechanical responses and the elevated instability in the spine has been known to affect the intervertebral disc pressure and the muscular activity level and finally result in back pain or LBDs (Solomonow, 2004).

The 10 minute recovery after the prolonged stooping was not enough for the full recovery of the creep of full lumbar flexion angle and this was consistent with earlier studies. This slow recovery might be due to the repetitive full flexion and lifting tasks that occurred every 2.5 minutes during the recovery phase (McGill and Brown, 1992), but a study with the spine of feline model also showed that creep by 10 minute flexion was not fully recovered by 10 minute rest period and large cumulative creep was observed at the end of cycles of flexion-rest period (Solomonow, Baratta, Zhou, Burger, Zieske, & Gedalia, 2003). The normalized EMG of extensor muscles showed faster recovery than the full flexion angle and this might be related to the reduced EMG and force-generating capacity of back extensors that have been observed after passive stretch of muscles (Fowles, Sale, & MacDougall, 2000). Together with strained passive tissues, the reduced mechanical capacity of the back extensors could reduce the safety margin in the lumbar spine and finally result in serious damage in the low back, especially when high exertion is required right after prolonged stooped posture such as standing up or lifting a weight from fully flexed posture or seated posture. A 30-second rest break moderated the creep of the full lumbar flexion angle and this showed the benefit of the micro-break during prolonged stooping tasks as it could reduce the cumulative strain of passive tissues in prolonged stooping. But the benefit of the break to the muscle activity level was questionable as a prolonged significant difference in the EMG between the two rest conditions was not observed.

The FE model simulated a prolonged stooping and upright standing posture with cyclic weight holding tasks, and the predicted results were compared with experimental observations to validate the model. A good agreement in the full lumbar flexion angle between the FE model simulation and in-vivo experiments confirmed the validity of the FE model in simulating the stooping posture. Less strong influence of the rest break in the FE simulation could be attributed to a modeling assumption that the gain of strength of passive tissues at recovery would follow the inverse path of the stress-relaxation curve. Based on the comparison with in-vivo data, it was believed that in-vivo lumbar spine tissues recover their strength faster than the FE model in the earlier stage of the recovery period.

In the interpretation of the quantified results of the force prediction, it should be noted that the current FE model included only major lumbar extensors and neglected the function of other muscles and tissues such as latissimus dorsi and lumbodorsal fascia which were known to be involved in extension moment generation and lumbar stabilization (Bogduk, Johnson, & Spalding, 1998; Gracovetsky, Farfan, & Lamy, 1981). Even though the contribution of those tissues in the lumbar spine is relatively small or doubtful (McGill, 2002) compared to the major extensor muscles, the future FE model may need to consider the function of other tissues. This is also important to understand the co-activation of muscles in the lumbar spine region in the change of extensor muscle capacity after passive stretching. Despite these limitations and assumptions, the validation of the model by comparing its predicted results with experimental results indicated a good agreement in terms of mechanical behaviors in prolonged stooping. The results of this validation study confirmed the capability of this FE model as a tool to assess the physical risks of prolonged stooping.

## REFERENCES

- Adams, M.A., & Dolan, P. (1996). Time-dependent changes in the lumbar spine's resistance to bending. *Clinical Biomechanics*, 11, 194-200.
- Bogduk, N., Johnson, G., & Spalding, D. (1998). The morphology and biomechanics of latissimus dorsi. *Clinical Biomechanics*, 13, 377-385.
- Bogduk, N., Macintosh, J.E., & Pearcy, M.J. (1992). A universal model of the lumbar back muscles in the upright position. *Spine*, 17, 897-913.
- Fowles, J.R., Sale, D.G., & MacDougall, J.D. (2000). Reduced strength after passive stretch of human plantarflexors. *Journal of Applied Physiology*, 89, 1179-1188.
- Goel, V.K., Monroe, B.T., Gilbertson, L.G., & Brinckmann, P. (1995). Interlaminar shear stresses and laminae separation in a disc. *Spine*, 20, 689-698.
- Gracovetsky, S., Farfan, H.F., & Lamy, C. (1981). The mechanism of the lumbar spine. *Spine*, 6, 249-262.
- Kippers, V., & Parker, A.W. (1984). Posture related to myoelectric silence of erectors spinae during trunk flexion. *Spine*, 9, 740-745.
- Marchand, F. & Ahmed, A.M. (1990). Investigation of the laminate structure of lumbar disc annulus fibrosus. *Spine*, 15, 402-410.
- McGill, S.M., & Kippers, V. (1994). Transfer of loads between lumbar tissues during the flexion-relaxation phenomenon. *Spine*, 19, 2190-2196.
- McGill, S. (2002). Low back disorders. Evidence-based prevention and rehabilitation. Human Kinetics. Champaign, IL.
- McGill, S.M., & Brown, S. (1992). Creep responses of the lumbar spine to prolonged full flexion. *Clinical Biomechanics*, 7, 45-48.
- Montgomery, D. (2001). Design and Analysis of Experiments (5th Ed.). New York: John Wiley & Sons.
- Panjabi, M.M., Goel, V., Oxland, T., Takata, K., Duranceau, J., Krag, M., & Price, M. (1992). Human lumbar vertebrae. Quantitative three-dimensional anatomy. *Spine*, 17, 299-306.
- Shirazi-Adl, A., Ahmed, A. M., & Shrivastava, S. C. (1986). A finite element study of a lumbar motion segment subjected to pure sagittal plane moments. *Journal of Biomechanics*, 19, 331-350.
- Solomonow, M. (2004). Ligaments: a source of work-related musculoskeletal disorders. *Journal of Electromyography and Kinesiology*, 14, 49-60.
- Solomonow, M., Baratta, R.V., Zhou, B.H., Burger, E., Zieske, A., & Gedalia, A. (2003). Muscular dysfunction elicited by creep of lumbar viscoelastic tissue. *Journal of Electromyography and Kinesiology*, 13, 381-396.
- Wang, J.L., Parnianpour, M., Shirazi-Adl, A., Engin, A.E., Li, S., & Patwardhan, A. (1997). Development and validation of a viscoelastic finite element model of an L2/L3 motion segment. *Theoretical and Applied Fracture Mechanics*, 28, 81-93.
- Winter, D. (2005). Biomechanics and motor control of human movement. John Wiley & Sons, 3<sup>rd</sup> ed. Hoboken, NJ.