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Viscoelastic Response of the Human Lower Back to Passive Flexion: The Effects of Age

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Abstract—Low back pain is a leading cause of disability in the elderly. The potential role of spinal instability in increasing risk of low back pain with aging was indirectly investigated *via* assessment of age-related differences in viscoelastic response of lower back to passive deformation. The passive deformation tests were conducted in upright standing posture to account for the effects of gravity load and corresponding internal tissues responses on the lower back viscoelastic response. Average bending stiffness, viscoelastic relaxation, and dissipated energy were quantified to characterize viscoelastic response of the lower back. Larger average bending stiffness, viscoelastic relaxation and dissipated energy were observed among older vs. younger participants. Furthermore, average bending stiffness of the lower back was found to be the highest around the neutral standing posture and to decrease with increasing the lower back flexion angle. Larger bending stiffness of the lower back at flexion angles where passive contribution of lower back tissues to its bending stiffness was minimal (i.e., around neutral standing posture) highlighted the important role of active vs. passive contribution of tissues to lower back bending stiffness and spinal stability. As a whole our results suggested that a diminishing contribution of passive and volitional active subsystems to spinal stability may not be a reason for higher severity of low back pain in older population. The role of other contributing elements to spinal stability (e.g., active reflexive) as well as equilibrium-based parameters (e.g., compression and shear forces under various activities) in increasing severity of low back pain with aging should be investigated in future.

Keywords—Age-related differences, Lower back bending stiffness, Passive deformation, Viscoelastic response.

INTRODUCTION

Low back pain (LBP) remains a major public health problem with considerable economical cost.[13,26] Low back pain usually initiates in early life with the most recurrence incidences between 35 and 55 years of age.[8,9,23,30] However, the severity of the problem, sickness and duration of symptom increase with age[8,9,23,30] such that the complaints of pain vary from ~45 to 85% in the elderly to ~15 to 30% in the general population.[10] Among older individuals, particularly those older than 65 years, LBP has been suggested to be a leading cause for chronic health problems.[19] Given that the population of most countries is aging and an increasing number of older individuals is remaining in the workforce, it is expected that the significance of LBP among older population to further increase in coming years.[46] Accordingly, it is substantially important to investigate the underlying mechanisms responsible for higher severity of LBP among older individuals.

Spinal instability has been suggested to play an important role in occurrence of LBP.[15,35] Spinal instability is clinically diagnosed by observation of relatively large intervertebral angular/translational motion while performing normal range-of-motion activities.[7,14,24] Biomechanically, though, spinal instability may be defined as the failure of the lower back neuro-musculoskeletal system to maintain the spine around its equilibrium condition (static or dynamic) following a perturbation.[28] Three subsystems of the lower back musculoskeletal system are coordinated to provide biomechanical stability to the very unstable spinal column. These include (1) the passive subsystem, involving the ligamentous spine and passive muscle forces, (2) the active subsystem (i.e., volitional and reflexive muscle forces), and (3) the neural control

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subsystem.[34] Structure, composition and behavior of tissues constituting these musculoskeletal subsystems change with aging. For instance, active muscular strength decreases during the aging process that has been attributed to the reduction in muscle mass and cross sectional area.[16] Similarly, age-related changes in lumbar intervertebral discs have been suggested to be due to progressive loss of tissue structure with aging.[1,11,33,37,50] Despite such knowledge about the age-related changes in tissues of musculoskeletal subsystems that stabilize the spine, it is not clear how contribution of these subsystems to spinal stability change with age.

Passive deformation tests have been used in the past to obtain an indirect estimate of spinal stability by characterizing the system's resistance to the applied deformation (i.e., a measure of bending stiffness). Most of earlier studies that used passive deformation tests were conducted on the osteoligamentous lumbar spine.[3,29,47] Results from these studies suggested that there is a deformation region around the neutral posture of the testing specimen that is associated with minimal resistance to passive deformation and hence very unstable. Such a region was however absent in results from *in vivo* passive deformation tests that were conducted on the whole lower back (i.e., lumbar spine and the surrounding tissues).[31] Comparison of results obtained from studies on the whole lower back vs. on osteoligamentous lumbar spine highlighted the stabilizing role of the lower back tissues surrounding the spine. A limitation of these *in vivo* studies, however, was that they were conducted in laydown position hence not accounting for the effects of gravity load and corresponding internal tissues responses on the lower back response to passive deformation.[27,31]

We have developed a new experimental tool for conduct of passive deformation test in upright standing posture, therefore, we are able to address the above mentioned shortcoming of earlier studies. In our design, passive deformation test in upright standing posture is achieved by inducing passive flexion to the lower back without changing trunk position through raising the legs. Such an approach minimizes the changes in active response of back muscles but, as opposed to laydown testing paradigm, maintain the effect of existing muscle activity in upright posture on our measures. Accordingly, the objective of present study was set to assess the age-related differences in the viscoelastic response of the lower back to passive flexion tests in upright posture. Viscoelastic response was quantified using measures of lower back average bending stiffness, the viscoelastic relaxation of lower back resistance, and the dissipated energy. If spinal instability is in part responsible for increase in the incidences of LBP with aging, a diminishing contribution to spine stability from subsystems that stabilize the spine (i.e., reflected in smaller value of bending stiffness) with aging should be expected. However, reports of reduced lower back range of deformation (e.g., flexion) with aging[39,41,43] as well as increased stiffness of intervertebral disc,[17,52] suggest a likelihood of larger bending stiffness for the lower back in the elderly. Alhough the reported reduction in range of lumbar flexion with aging could be due to an active muscle control in trunk motion, given the reported increase in stiffness of intervertebral disc with aging, we hypothesized that the lower back resistance to passive deformation in upright standing posture to be larger in older vs. younger individuals. Therefore, a potential deterioration of spinal stability due to reduced bending stiffness of the lower back with aging is unlikely.

METHOD

Participants

Five equal-sized and gender-balanced age groups (Table 1) were formed by recruiting sixty healthy individuals. The age groups divisions were intended to represent individuals in the 1st to 5th decades of working life. Prior to any data collection, all participants completed an informed consent procedure approved by the University of Kentucky Institutional Review Board. Inclusion criteria were, no recent history of LBP, lack of a history of working in physically demanding occupations, moderate levels of physical activity outside the workplace, and a BMI between 22 and 30 and. There was no significant differences in stature ($p = 0.851$), body mass ($p = 0.127$) or body mass index (BMI) ($p = 0.139$) between the five age groups (Table 1; verified using univariate Analysis of Variance-ANOVA).

Experimental Procedures

To enhance the reliability of our results, each participant completed two identical experimental sessions, which were conducted in the morning to minimize the influence of diurnal changes in the lower back biomechanics.[4] During each session, participants completed two passive flexion–extension and one active flexion–extension tests. The active flexion–extension test was conducted on the ground to obtain lower back range of flexion for each participant. During this test the participants were instructed to slowly (i.e., taking ~4 s) bend forward from upright standing posture to reach their maximum comfortable trunk flexion in the sagittal plane, to pause for five seconds in such a posture, and then to return slowly back to the original

TABLE 1. Mean (SD) participant characteristics.

	Age (years)		Stature (m)		Body mass (kg)		Body mass index	
Age group	M	F	M	F	M	F	M	F
22–28	25.6 (1.0)	23.5 (2.3)	177.8 (6.8)	164.9 (3.7)	78.5 (4.7)	61.4 (6.4)	24.9 (2.7)	22.5 (1.6)
32–38	33.5 (2.2)	34.0 (1.2)	173.0 (5.1)	167.4 (7.1)	81.3 (10.3)	64.5 (10.2)	27.1 (3.3)	22.9 (2.5)
42–48	44.5 (1.8)	45.1 (1.4)	179.9 (4.8)	166.2 (5.4)	88.0 (12.0)	70.1 (12.1)	27.2 (4.1)	25.3 (3.8)
52–58	54.3 (1.7)	56.0 (2.3)	180.5 (10.4)	163.4 (6.0)	85.4 (11.3)	72.0 (8.7)	26.1 (1.8)	26.3 (3.1)
62–68	65.6 (1.6)	65.0 (2.7)	179.7 (6.2)	163.5 (5.7)	86.3 (11.1)	61.0 (4.1)	26.6 (1.6)	22.8 (1.2)

Each age group included 12 gender-balanced participants.

upright standing posture. Participant were instructed to repeat the active flexion–extension tests three times while thorax and pelvis rotations were measured using two wireless Inertial Measurement Units (IMUs; Xsens Technologies, Enschede, Netherlands) that were attached to the T10 vertebral process and the sacrum (S1) using straps. From the active flexion–extension test, the subject's lower back range of flexion was calculated by subtracting the sacrum rotation from the T10 rotation at the time of the maximum T10 rotation. Passive flexion–extension tests were conducted using a custom-made rigid metal frame wherein motion of trunk was constrained at pelvis using straps and at thorax (T8 level) using a harness-connecting rod assembly which rigidly connected participant's thorax to a fixed point (Fig. 1). The passive flexion tests were completed within this frame by rotating participant's legs around their lower back (~S1) with an angular velocity of ~3°/s using an actuated leg platform (Fig. 1). Passive flexion–extension test started with participant in standing posture, followed by rotation of participants' leg to achieve the desired lumbar flexion, maintaining the participant at that posture for four minutes and then extending them back, with the same velocity, into the initial standing posture on the frame. Given that the lower extremities and the pelvis of participant were constrained to the leg platform and the center of rotation of the platform was aligned with the approximate location of the S1 in the sagittal plane, it was assumed that the amount of induced lower back flexion was the same as the amount of rotation of leg platform ($<2°$ error). During these experiments, the rotation of leg platform was visually controlled using a protractor attached to its axis of rotation and was measured using a wireless IMU (Fig. 1). Raising participant's legs to induce lower back flexion stretched their lower back tissues and hence caused a trunk extension exertion that was resisted by the harness-connecting rod assembly. Therefore, resistance of lower back tissues to passive flexion was indirectly measured using an inline load cell on the harness-connecting rod assembly (Interface SM2000, Scottsdale, AZ) (Fig. 1).The final lumbar flexion was set 40° for the first passive flexion–extension test and the 70% of participant's lower back range of flexion (obtained from active flexion–extension test) for the second passive flexion–extension test. Both a fixed (i.e., 40°) and a subject-specific (i.e., 70% of participant's lower back range of flexion) level of lower back flexion were considered to better understand the viscoelastic response of lower back to passive deformation with or without consideration of age and gender related differences in an individual flexibility.

All participant completed the passive flexion–extension at 40° lower back flexion, followed by active flexion–extension test, and finished by the passive flexion–extension at 70% of lower back range of flexion. A minimum of 10 min rest period was provided between each two consecutive tests to allow for viscoelastic recovery of the lower back following the first passive flexion test. The accelerometers data was sampled at 50 Hz; while the load cell data was collected at 3000 Hz. The raw kinetics and kinematics data were low-pass filtered using a fourth-order, bidirectional, Butterworth filter with a 50 Hz and a 6 Hz cutoff frequency, respectively. Electromyography activity of select back and abdominal muscles were monitored during the passive tests to assure their level of activity do not change. These muscles included the bilateral erector spinae at the L3 and the L5 levels, rectus abdominis, and external oblique (see[49] for details of electrodes placements).

Data Analysis

An in-house program was developed in MATLAB software (The MathWorks Inc., Natick, MA, USA, version 7.13) to characterize the viscoelastic response of lower back during the passive flexion tests by calculating the following outcome measures from each of the two tests (see below for details): (1) average bending stiffness over three flexion ranges (i.e., starting at the upright standing posture and ending at 12.5, 25, and 100% of the final passive flexion angles), (2) the amount of relaxation in the lower back resistance (i.e., quantified by a measure of moment) after four minutes

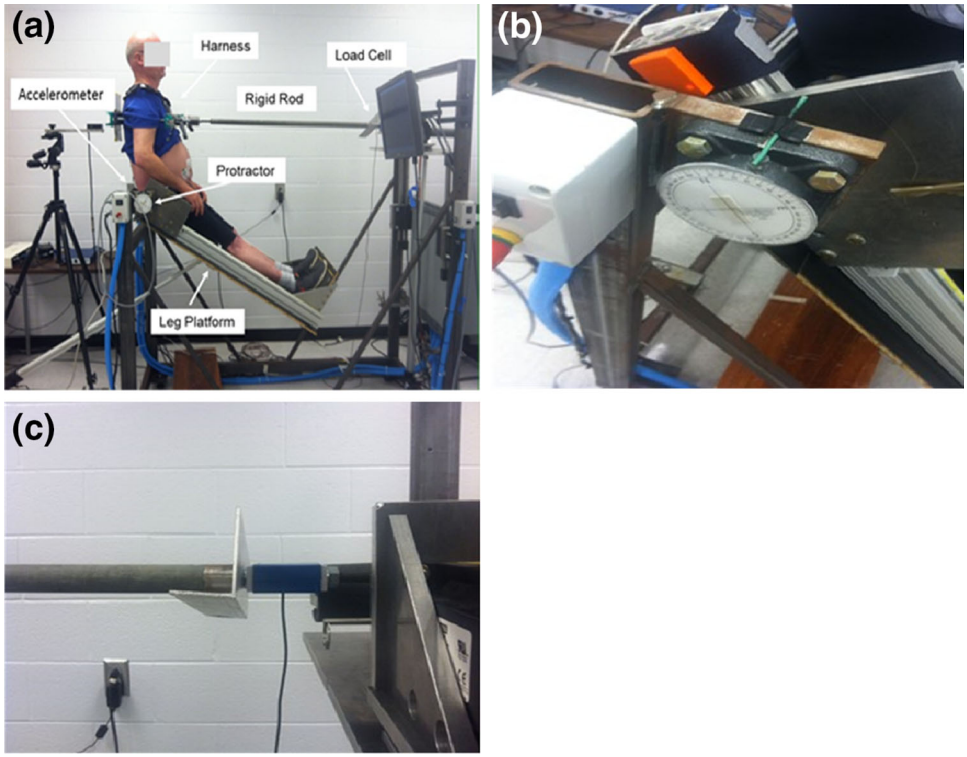

FIGURE 1. (a) Passive flexion test experimental setup; (b) the protractor and inertial measurement unit used to respectively control and measure angular rotation of leg platform; and (c) the load cell used to measure tension in the harness-connecting rod assembly.

of sustained flexed posture, (3) the amount of energy dissipated during the test, and (4) the viscoelastic state of lower back. To calculate these outcome measures six event time points (i.e., start point, 12.5, 25, and 100% of target point, extension point, and end point) were identified on the synchronized kinematics and kinetics data (Fig. 2). The start point was the earliest time in the collected kinematics data wherein angular velocity of the platform became greater than mean plus two standard deviation of kinematics data collected during one second prior to that time point. The target point was the first point after the start point with angular velocity of platform equal to zero and corresponded to the time of achieving the final desired lower back flexion. 12.5 and 25% of target point were the time points corresponded to the 12.5 and 25% of lower back target flexion respectively and were selected to investigate the presence of a neutral zone. The extension point corresponded with the start time of lowering participant's leg and was the first point after the target point wherein the angular velocity of platform became smaller than the mean minus two standard deviation of kinematics data collected during one second prior to that time point. Finally, the end point was defined as the first time point after the extension point wherein platform velocity became zero and which corresponded to the end of passive flexion test (Fig. 2). The average bending stiffness over each of the three flexion ranges was calculated using the following relationship

$$K_{\text{ave}} = (M_{\text{e}} - M_{\text{s}})/(\theta_{\text{e}} - \theta_{\text{s}})$$

where the M_{s} and θ_{s} were moment and lower back flexion at the standing start point, while M_{e} and θ_{e} were moment and lower back flexion at the end point of flexion range (i.e. at the time of 12.5, 25, and 100% of target point).

The moment at each time point was calculated by multiplying the measured force (i.e., *via* load cell) at that time point by the distance between the harness and the axis of rotation of platform (measured for each subject before the first passive flexion test). The relaxation moment (M) was defined as the difference in moment values between the target and extension points. The dissipated energy (E) during the test was calculated as the area inside the closed moment- lower back flexion curve (Fig. 2b). Finally, the viscoelastic state was defined as the ratio of dissipated energy (E) to input energy (E_{input}), where input energy was the stored energy (i.e., strain energy) during the flexion phase.[25,51]

For each participant and dependent measure, the mean value across two sessions was used for statistical analyses. A mixed-factors ANOVA was used to assess the effects of age and gender, as between-subject vari-

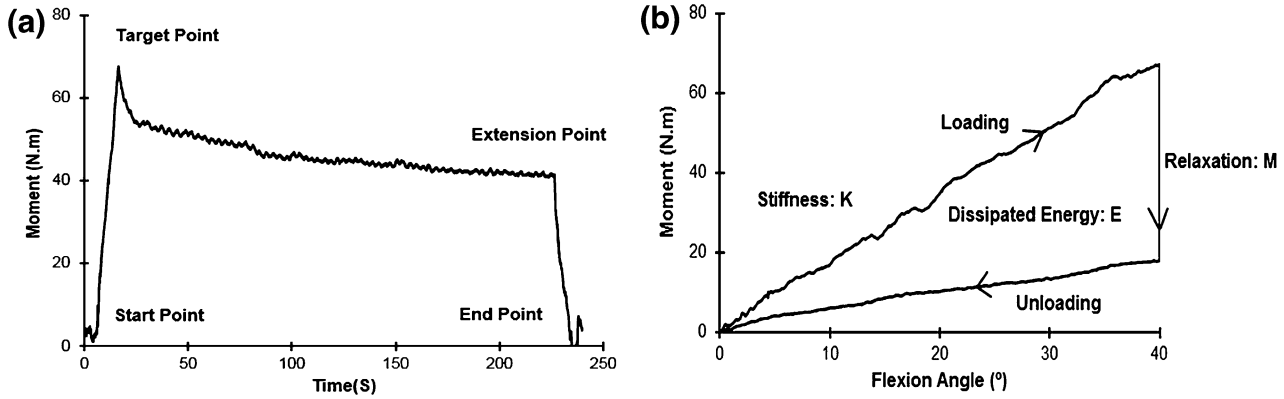

FIGURE 2. Sample recorded lower back resistance (i.e., quantified by a measure of moment) as (a) a function of time; and (b) a function of lower back flexion during the passive flexion tests; (b) indicates the lower back flexion up to the target point shown in (a).

ables, and the amount of flexion range, as the within-subject variable, on the lower back average bending stiffness. For the other dependent measures, a univariate two-way ANOVA with age and gender as the independent variables was performed. Significant univariate ANOVAs were followed by *post hoc* analyses using Tukey's procedure. Since the two passive flexion tests were independently designed, separate statistical analyses were performed for each test. All statistical analyses were performed using SAS (version 9.4, Dell Inc), and summary values are reported as means (SD). In all cases, a p value ≤ 0.05 was considered as statistically significant.

RESULTS

Passive Flexion–Extension-1 (Target Lumbar Flexion: 40°)

The average bending stiffness of lower back depended on the age ($F = 4.88$, $p = 0.002$), gender ($F = 21.18$, $p < 0.001$), and flexion range ($F = 29.76$, $p < 0.001$) as well as the interaction between gender and flexion angle ($F = 4.21$, $p = 0.017$). The bending stiffness was larger in older vs. younger population, reduced with increasing flexion range, and was smaller in female vs. male participants regardless of the flexion range (Fig. 3). Specifically, mean (SD) of average bending stiffness was 79 (36) Nm/rad vs. 136 (68) Nm/rad in female vs. male participants. The amount of dissipated energy was larger in older vs. younger ($F = 3.99$, $p = 0.007$) and in male vs. female ($F = 16.30$, $p < 0.001$) participants. Means (SD) of dissipated energy for all age groups are given in Fig. 4 while their respective values for male and female participants were 16(6) and 9(4) Nm rad. The relaxation moment was similarly larger in older vs. younger ($F = 3.19$, $p = 0.021$) and in male vs. female ($F = 11.05$, $p = 0.002$) participants. Means (SD) of relaxation moment for all age groups are given in Fig. 4 while their respective values for male and female participants were 40(15) Nm vs. 25(11) Nm. Finally, neither age ($F = 0.85$, $p = 0.501$) nor gender was found to affect viscoelastic state with mean (SD) ratio of 0.48(0.18).

Passive Flexion–Extension-2 (Target Lumbar Flexion: 70% of Lower Back Range of Flexion)

Similar to passive flexion at 40° lower back flexion, age ($F = 5.08$, $p = 0.002$), gender ($F = 20.21$, $p < 0.001$), and flexion range ($F = 53.07$, $p < 0.001$) as well as the interaction between gender and flexion angle ($F = 6.231$, $p = 0.003$) had significant effects on the average bending stiffness of lower back. The bending stiffness was larger in older vs. younger participants (Fig. 5) and in male vs. female participants (129 (60) vs. 75 (31) Nm/rad). The average bending stiffness reduced with increasing flexion range (Fig. 5) and was, in general, smaller in female vs. male participants (Fig. 5). While there was no significant differences in final lower back flexion representing 70% of lower back flexion between age groups (42(8)°), the lower back flexion angles were significantly larger ($F = 12.59$, $p < 0.001$) among male (46(7)°) vs. female (38(8)°) participants. All age-related differences in viscoelastic response of the lower back to passive deformation during this test were consistent with those obtained from passive flexion test at 40° including dissipated energy: $F = 2.75$, $p = 0.038$; relaxation moment: $F = 3.28$, $p = 0.019$ (Fig. 6); and the viscoelastic state: $F = 0.72$, $p = 0.617$.

Similar to passive flexion at 40° lower back flexion, the dissipated energy was larger ($F = 24.69$, $p < 0.001$) among male vs. female participants, at respectively 16(7) vs. 6(3) Nm rad during passive flexion at 70% of

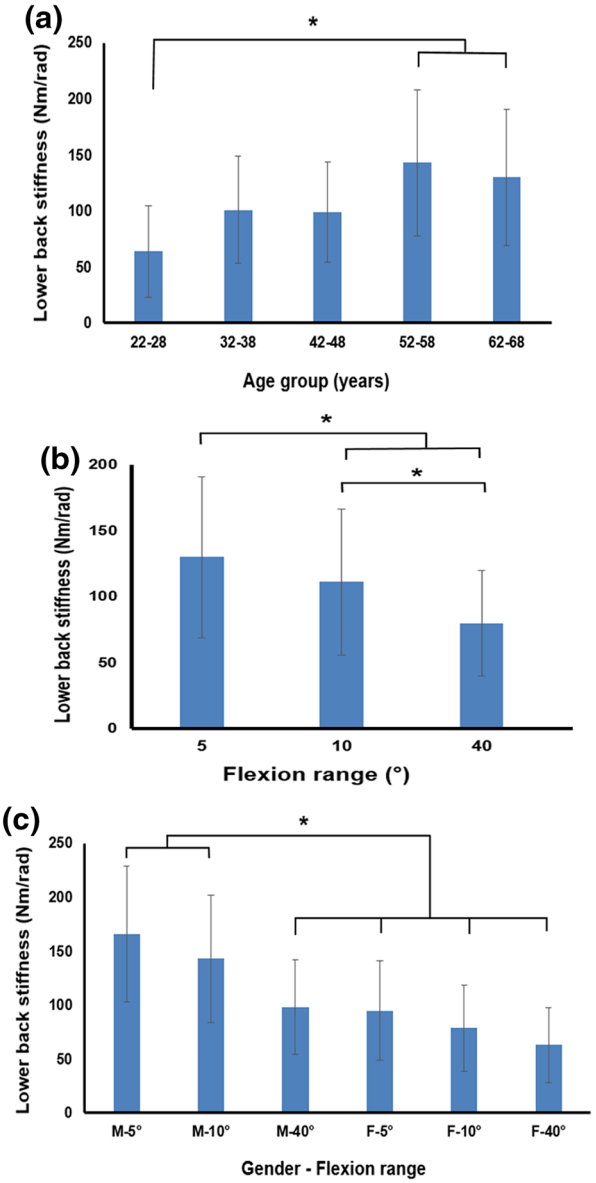

FIGURE 3. The effect of: (a) age; (b) flexion range; and (c) gender (M: Males and F: Females) by flexion range on the lower back average bending stiffness during passive flexion at 40° lower back flexion. Significant differences between age groups are indicated with brackets.

lower back range of flexion. Relaxation moment was also larger ($F = 15.72$, $p < 0.001$) among male vs. female participants (34 (15) vs. 16 (9) Nm). Finally, there was no differences in viscoelastic state between male and female participants with mean (SD) ratio of 0.44 (0.20).

DISCUSSION

The main objective of this study was to assess potential age-related differences in the viscoelastic response of lower back to passive flexion in upright standing posture. While there was no differences in the measure of viscoelastic state between age and gender groups, larger average bending stiffness (i.e., confirming our hypothesis), relaxation and dissipated energy were observed among older vs. younger participants. During both passive flexion tests, most significant differences in outcome measures were observed between individuals older than 50 years and younger than 30 years.

The larger lower back average bending stiffness of older vs. younger participants found in our study is in agreement with earlier reports of reduced lower back range of flexion and increase stiffness of intervertebral discs with aging.[17,39,41,43,52] Our results suggest that the reduction in range of motion with aging is more likely to be due to increased bending stiffness of the lower back than active control of motion. Increase of lower back resistance to passive flexion in upright standing with aging suggest that a diminishing contribution of passive and volitional active subsystems to spinal stability may not be a reason for higher severity of LBP in older population. This suggestion is further supported by our recent finding of comparable values of trunk intrinsic stiffness (i.e., a measure primarily reflecting the active volitional contribution of trunk musculoskeletal system to spinal stability) among younger and older individuals.[49] Increase in the lower back bending stiffness with aging, while positively affecting spinal stability, have been suggested to compromise the postural control where precise motor control (i.e., during balance, standing, gait, etc.) is required, therefore leading to increase risk of falling.[21,22,36,48] Such opposing effects of increase in the lower back bending stiffness on spinal stability and whole body balance call for different level of involvement from the active reflexive trunk response. Specifically, with an increase in the lower back bending stiffness, stabilizing the spine would require less contribution from the reflexive trunk response whereas keeping whole body balance would require more contribution from the reflexive trunk response. Whether age-related changes in reflexive behavior of trunk are according to less spine stabilizing demand or more whole body balance requirement should, however, be determined in future.

The amount of moment drop during relaxation periods was larger among older vs. younger participants (Figs. 4, 6). Larger relaxation in the elderly may likely be due to the reduction in muscle mass and cross sectional area and an increase in fatty infiltration with aging.[16] The relaxation of 43(27) %, observed in the 22-28 age group of our study, was comparable with the reported value of 41(22) % by Toosizadeh *et al.*[45] which was also obtained from a relatively young participant cohort. Earlier *in vitro* passive deformation

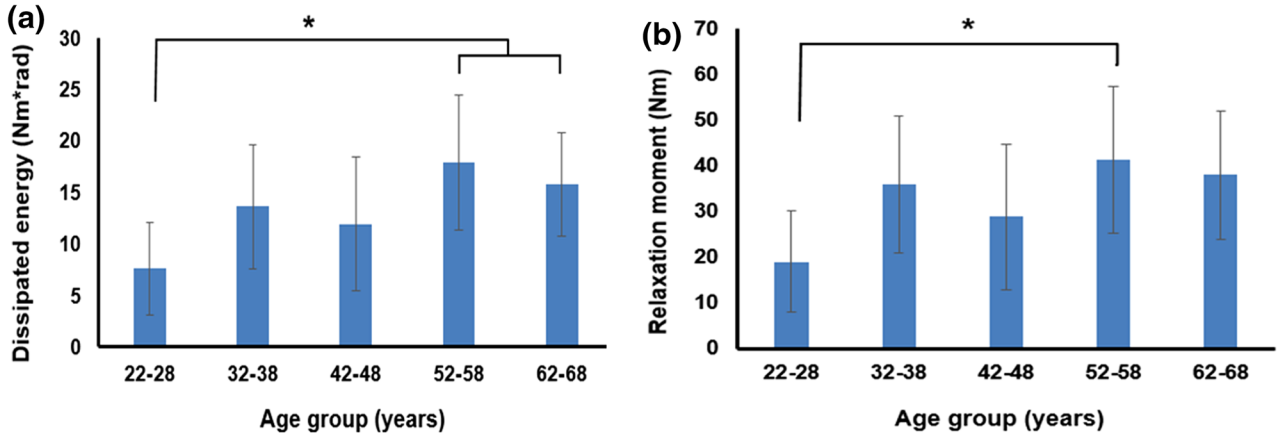

FIGURE 4. Age-related differences in: (a) dissipated energy; and (b) relaxation moment for passive flexion at 40° lower back flexion. Significant differences between age groups are indicated with brackets.

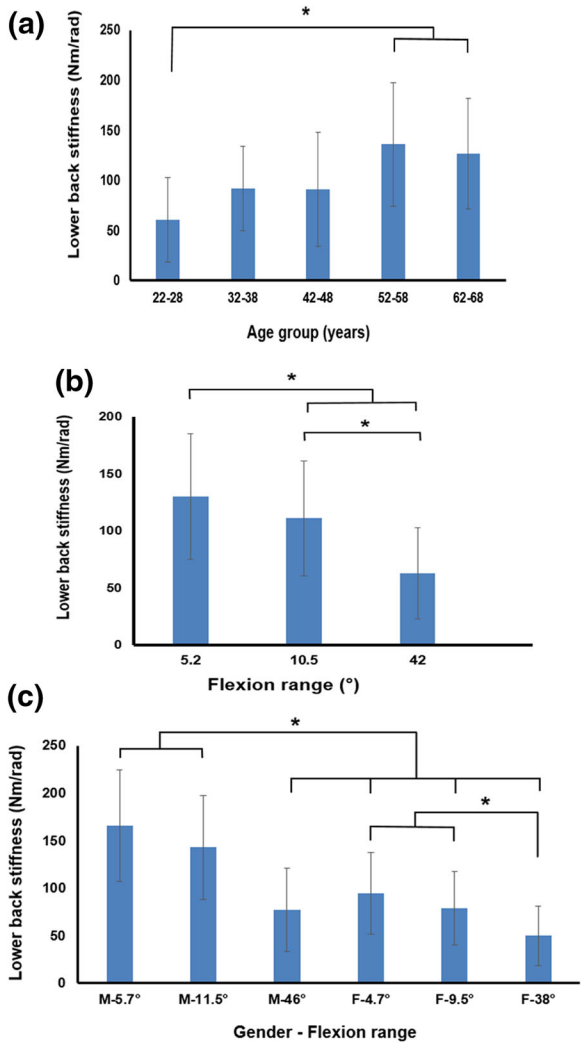

FIGURE 5. The effect of: (a) age; (b) flexion range; and (c) gender (M: Males and F: Females) by flexion range on the lower back average bending stiffness during passive flexion at 70% of lower back range of flexion. Significant differences between age groups are indicated with brackets.

studies have reported relaxations of ~48% for osteoligamentous lumbar spine motion segments[3] and ~27% for muscles.[6,38] Reduced lower back bending stiffness following viscoelastic relaxation has been suggested to be associated with diminished spinal stability and a higher risk for LBP.[20,44] Although average bending stiffness was larger among older vs. younger participants, the corresponded larger amount of moment drop during the relaxation period resulted in comparable final bending stiffness between different age groups. Therefore, regardless of age, risk of spinal instability due to reduced trunk bending stiffness is equally elevated following viscoelastic relaxation of the lower back. Further, viscoelastic changes in the mechanical behavior of spinal ligaments can lead to sensory motor disturbances.[42] Creep deformation of spinal ligaments has been recognized to be associated with delayed and reduced stretch-reflex response of trunk muscles, which plays an important role in providing spinal stability.[32] Therefore, it is also important to investigate age-related differences in disturbances to reflexive behavior of trunk neuromuscular system following viscoelastic changes in the mechanical behavior of spinal ligaments.[5]

A viscoelastic state ratio equal to zero represents a pure elastic material while a ratio equal to one describes a pure viscos material.[25,51] Viscoelastic state ratios between 0.1 and 0.59 for spinal ligaments,[3] and equal to 0.2 for intervertebral discs under axial force[18] have been reported in load-relaxation tests. Toosizadeh *et al.*[45] reported no changes in viscoelastic state (mean (SD) ratio = 0.42 (0.15)) with changes in lower back flexion angles among a group of young individuals. We also did not find any significant differences in viscoelastic state between age and gender groups indicating that an increase in lower back bending stiffness (subsequently an increase in input

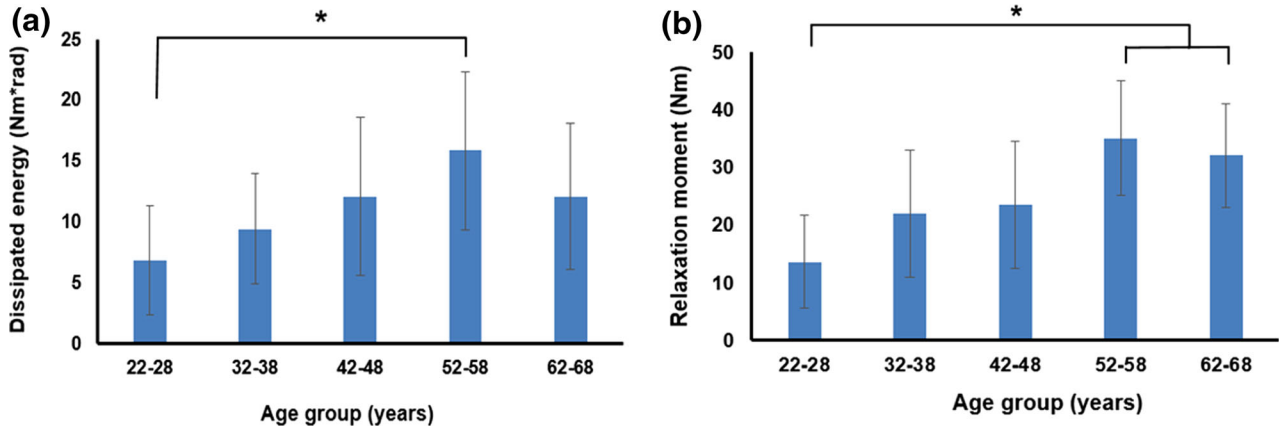

FIGURE 6. Age-related differences in: (a) dissipated energy; and (b) relaxation moment. Significant differences between age groups are indicated with brackets.

energy E_{input}) is associated with larger dissipated energy and vice versa such that the overall viscoelastic state remains unchanged between age groups. Such a finding, in combination with other outcome measures, suggests that while the immediate mechanical response of lower back to deformation is different between age and gender groups, its prolonged mechanical response is similar.

McGill *et al.*[31] showed that in a laydown posture the lower back resisted the passive flexion from the beginning of motion (i.e. no neutral zone) and the bending stiffness in all points was higher than those reported for the osteoligamentous spine.[2] Consistent with McGill *et al.*,[31] our results indicated that in a standing posture, where the active trunk muscles are present, the lower back resisted the flexion from the beginning of motion such that no neutral region with small bending stiffness was present. Moreover we found that lower back bending stiffness to be larger around the neutral posture than other flexed postures. Given that the resistance of lumbar spine to flexion as well as passive resistance of trunk muscles and spinal ligaments around neutral posture is minimal, our results suggested that the bending stiffness in standing neutral posture was primarily governed by the required active muscle responses to hold the posture. This suggestion concurs with results from Cholewicki *et al.*[12] wherein using modeling and experimental procedures demonstrated that the minimal activity of trunk flexor–extensor muscles around the neutral standing posture provided the required mechanical stability to the lower back in healthy individuals.

To increase our ability to assess the hypotheses and draw robust conclusions about the mechanics of lower back and the underlying mechanisms responsible for LBP, rather restrictive inclusion/exclusion criteria were considered. Such criteria, though, reduces the generalizability of the results to some extent. The current tests and methods can be used in future studies with population exposed to a broader range of occupational risk factors. The velocity of lower back flexion in our passive deformation test was ~3°/s which may have been slower than the pace of a real life flexion task. However, such a velocity was adopted to avoid any potential tissue injury. Although the muscle activity remained constant (i.e., an average change of $<0.15\%$ of activity at maximum voluntary exertion for both back and abdominal muscles) during the passive deformation tests, alterations in muscle length can change their output force and, in turn, the estimated bending stiffness at the target point. Given the small level of activity in all trunk muscles (i.e., $<2.2\%$ of activity at maximum exertion), alterations in muscle force with changes in their length during passive deformation tests were expected to have minimal impact on our estimates of bending stiffness.

In conclusion, while the severity of LBP is much higher in older vs. younger population, spinal instability, due to diminishing collective contributions from active volitional and passive lower back tissues, may be a less likely contributing factor. The role of other contributing elements to spinal stability (e.g., active reflexive) as well as equilibrium-based parameters (e.g., compression and shear forces under various activities) should be investigated in future. Spinal instability could, however, be a determining factor in LBP among females when considering the significantly smaller bending stiffness of lower back around neutral standing posture in females vs. males. Given such smaller bending stiffness of lower back in females, the reported smaller lower back range of flexion in females vs. males,[41] as opposed to the similar reported differences between older and younger individuals,[39,41,43] could potentially be due to an active protecting control of trunk motion (rather than larger bending stiffness). Age and gender-related differences in viscoelastic

response of lower back to passive flexion, reported here, can also be useful for biomechanical modeling effort.[40] Such biomechanical models can serve as effective tools for design and planning programs aimed at reducing LBP incidences.

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