Occupational Trunk Flexion and Neuromuscular Disturbance

Sponsor
National Institute for Occupational Safety and Health (NIOSH)

Grant #
5R01 OH008504

Performance Period
August 1, 2006 – July 31, 2012*

*includes a 1-year no-cost extension

Final Report

by

Maury A. Nussbaum, Ph.D. (PI)*
Michael L. Madigan, Ph.D. (Co-I)
Kevin Granata (deceased)

*Department of Industrial and Systems Engineering
Virginia Tech
250 Durham Hall (0118)
Blacksburg, VA 24061
nussbaum@vt.edu
(540) 231-6053

Awardee Institution:
Virginia Tech
Office of Sponsored Programs
1880 Pratt Drive, Suite 2006
Blacksburg, VA 24061

October 17, 2012

Table of Contents

ABSTRACT

Occupational tasks involving trunk flexion are associated with an increased risk for low back pain (LBP). Previous studies on humans or animal models suggest that prolonged or repetitive trunk flexion alters the viscoelastic behavior of trunk tissues. Recent studies on animal models, however, indicate that prolonged or repetitive stretching of tissues due to flexion also, and perhaps more importantly, alters trunk active neuromuscular behaviors (i.e., muscle reflexes). Considering these changes as disturbances from normal behavior, we aimed to quantify the effects of several distinct exposures to prolonged and repetitive trunk flexion on such disturbances and to measure recovery from them. A biomechanical model was developed to quantify changes in trunk passive and active behaviors, using measures of trunk mechanical impedance (i.e., apparent mass, stiffness, and damping) and reflexive neuromuscular responses. Experimental data were obtained from sudden position-perturbation experiments.

Exposures included: 1) prolonged flexed postures with and without added loads, 2) repetitive trunk flexion, and 3) repetitive lifting. Following prolonged trunk flexion, acute changes in trunk stiffness and reflexive responses were found, and which increased with both task duration and an external load. Though the acute disturbance to reflexive responses appeared to compensate for disturbances (decreases) in trunk stiffness, recovery to pre-exposure values occurred quickly. As such, this may leave the spine vulnerable to instability during the relatively slow recovery of trunk stiffness. In contrast, when the effects of trunk weight were excluded during the experiment, by raising participant's legs to induce flexion with the torso upright, the disturbance in reflexive responses continued even after full recovery of trunk stiffness. For this latter experiment, acute changes in trunk stiffness and reflexive response were larger with increasing trunk flexion angle, but similar between trunk flexion durations.

Repetitive exposures to trunk flexion that involved longer flexion durations and higher flexion duty cycles were both associated with increased disturbances in trunk stiffness and reflexive responses, and both remained unrecovered following 20 minutes of rest. Disturbances in reflexive trunk responses following repetitive exposures, in contrast with those during prolonged static flexed postures, did not appear as compensatory responses to disturbed (reduced) stiffness. Similar slow recovery patterns in trunk stiffness and reflexive responses were observed following exposures to repetitive lifting. Following repetitive lifting, though, an increasing disturbance was found only with increasing trunk flexion angles.

Other measures of trunk mechanical impedance (i.e., apparent mass) and reflexive behavior (i.e., reflex delay) were not substantially affected by any of the exposures. Both trunk stiffness and reflexive responses play important roles in stabilizing the spine. Hence, from a mechanical perspective, these results suggest a higher risk of injury due to spinal instability following exposure to tasks involving prolonged or repetitive flexed postures. In particular, persistent and simultaneous disturbances in trunk stiffness and reflexive responses, along with insufficient recovery time, are likely associated with a higher risk of spinal instability. The current work helps to understand the linkages between occupational trunk flexion and low back pain development, and may contribute to future ergonomic guidelines.

SIGNIFICANT (KEY) FINDINGS

Prolonged Trunk Flexion

Exposure to prolonged flexed trunk postures resulted in substantial acute changes in trunk stiffness and muscle reflexive responses. When raising the legs to induce trunk flexion, with no gravitational effects, acute changes in stiffness and reflexive behavior were larger following exposure to increasing trunk flexion angle, and with the largest changes occurring after maximum trunk flexion. When bending forward at the waist, acute changes in trunk stiffness and reflexive responses were larger following exposure to increasing trunk flexion duration. The presence of a small external load placed in the hands during flexion exposures caused larger acute changes only in trunk stiffness.

Recovery of these changes was dependent on the type of flexion posture. In Study 1, involving viscoelastic creep, recovery of trunk stiffness required roughly the same duration as the exposure, while trunk reflex responses remained elevated one hour after exposure to maximum trunk flexion. In Study 2, involving load relaxation, recovery of trunk stiffness was directly related to the exposure duration, requiring more time than initial exposure duration following longer flexion durations. In contrast to Study 1, however, trunk reflex responses did not remain elevated throughout the recovery period.

Repetitive Trunk Flexion

Creep deformations accumulated across all combinations of tested trunk flexion duration and duty cycle, suggesting insufficient recovery time between bouts of flexed postures. Substantial disturbances in trunk stiffness and reflexive responses occurred following 48 minutes of repeated trunk flexion. A longer duration of static trunk flexion performed with a 50% duty cycle resulted in the largest decrement in trunk stiffness. These disturbances were both in the same direction (i.e., decreasing from the normal state) and remained unrecovered following 20 minutes of standing recovery.

Repetitive Lifting

Changes in trunk stiffness and reflexive responses were more substantial for repetitive lifts performed with increased trunk flexion angle. Similar to repetitive trunk flexion, disturbances in stiffness and reflex responses were in the same direction and neither fully recovered after 20 minutes. However, the recovery rate of trunk stiffness was faster than trunk reflexive responses.

TRANSLATION OF FINDINGS

Consistent with epidemiological evidence of an association between flexed working postures and an increased risk of occupational LBP occurrence, dose-response relationships were found here between different aspects of exposure to flexed working posture and changes in several measures of trunk mechanical and neuromuscular behaviors. Specifically, larger trunk flexion angles, longer trunk flexion durations, or more frequent flexion, all led to larger disturbances in trunk behaviors and more prolonged recovery times. In particular, aspects of trunk behaviors (i.e., trunk stiffness and reflexive response) were investigated that can lead or contribute to LBP risk via mechanical injury. Mechanical injury occurs when a tissue is subjected to loads (stresses or strains) beyond its strength threshold. Changes in trunk stiffness and reflexive behaviors that were quantified here both suggest reductions in trunk tissue strength thresholds and potential increases in the loads induced on trunk tissues during activities involving trunk flexion. The latter changes in tissue loads should be quantified, in future research, by quantification of spinal loads and stability.

Within this mechanical injury conceptual context, our results suggest that occupational tasks requiring prolonged and/or repetitive postures, particularly those at or near full trunk flexion, expose workers to an increased risk for low back injury. This elevated risk is present not only during and immediately after trunk flexion, but also for extended durations following exposure. As such, trunk flexion duration and extent (angle) should be minimized through ergonomic (re)design, and recovery periods should be provided following trunk flexion.

OUTCOMES / IMPACT

Potential outcomes

- Acute changes in trunk stiffness and reflexive responses were dependent on trunk posture and flexion duration, and resulted in substantial disturbances for extended periods following flexion exposures. Therefore, controlling trunk posture and reducing the duration of flexion exposure are considered important ergonomic targets for minimizing low back injury risks and for minimizing carry-over effects from previous to subsequent job tasks.

- Increasing the duration of rest between trunk flexion events (within a given task) may also help minimize disturbances to trunk stiffness and reflexive responses. In particular, longer rest periods between consecutive bouts of trunk flexion (i.e., lower flexion duty cycle) may reduce the rate of disturbance accumulation. Where this is not practically feasible, however, controlling for posture, or rotating between tasks not requiring full trunk flexion, may be effective for reducing disturbances to trunk behaviors and subsequent low back injury risk.

- When performing tasks requiring repetitive lifting, the vertical position of the load should be brought closer to the waist to reduce the magnitude of trunk flexion during lifting tasks, and subsequent disturbances to trunk behaviors.

SCIENTIFIC REPORT

1. Background

Low-Back Disorders (LBDs) are the most prevalent source of musculoskeletal disability and one of the most common musculoskeletal problems in the United States (BLS, 1990; 1994). The risk of a LBD is associated with occupationally-related lifting (Andersson, 1981; Kelsey & White, 1980; Waters et al., 1999) and attributed in part to excessive spinal load and/or insufficient spinal stability (Chaffin & Park, 1973; Herrin et al. 1996; Magora, 1970; Manning et al., 1984; Manning & Shannon, 1981; Norman et al. 1998; Omino & Hayashi, 1992). LBD risk is high for individuals working in static flexed postures (Punnett et al., 1991) or tasks requiring repetitive dynamic lifting (Marras et al., 1993). Data from animal models suggest that this risk may be attributed, in part, to flexion-induced accumulation of neuromuscular disturbances that influence control of subsequent tasks (Solomonow et al., 1999). Recent evidence reveals similar neuromuscular disturbances following flexion tasks in humans (Granata et al., 2004; Moorehouse & Granata, 2005). Disturbances to neuromuscular control are important, because trunk muscle recruitment (co-contraction), trunk muscle reflex response, and active muscle stiffness contribute to spinal load and stability (Gardner-Morse et al., 1995; Granata & Marras, 1995; 2000; Hodges & Richardson, 1996; Radebold et al., 2000). Therefore, it is necessary to quantify the accumulation and recovery of neuromuscular disturbances caused by occupational flexion tasks.

Back Injuries and Flexed Working Postures

Back injuries in industry are common, costly, and debilitating. Musculoskeletal injuries continue to be the leading cause of worker disability (BLS, 1990; 1994). Among these, LBDs are the most prevalent source of musculoskeletal disability, and have been described as one of the most common and significant musculoskeletal problems in the United States (BLS, 1998). LBDs affect 59 - 80% of the population sometime in their lives (Kelsey et al., 1984), leading to substantial morbidity, disability and economic loss (Hollbrook et al., 1994; Praemer et al., 1992). Among people under 45 years of age, LBDs are the leading cause of activity limitation, affect up to 47% of workers with physically-demanding jobs (Andersson, 1981; Rowe, 1971), and account for 25% of all lost work days in the United States (Guo et al., 1995). About one-fifth of all workplace injuries and illnesses are back injuries, responsible for up to 40% of compensation costs (Spengler et al., 1986). The total costs associated with LBDs are substantial, with estimates of total societal costs from $25 to $100 billion annually (Cats-Baril & Frymoyer, 1991). Reflecting these data, the National Occupational Research Agenda (NORA) places control of LBDs as a priority area for intervention and research (Rosenstock et al., 1998).

Risk of LBD is associated with industrial lifting tasks (Andersson, 1981). Roughly one-third of occupational injuries in the United States are caused by overexertion, lifting, throwing, holding, carrying, pushing, and or pulling objects that weigh 50 lbs or less (NSC, 1989). The type of occupational work is closely related to the risk of suffering a LBD (Andersson, 1981b; Pope, 1989). In particular, manual materials handling (MMH), specifically lifting, dominate occupationally-related LBD risk. Retrospective studies of industrial injuries have identified MMH as the most common cause of LBD, estimating that lifting and MMH account for 50 - 75% of all back injuries (Bigos et al., 1986; Spengler et al., 1986).

Flexed working postures contribute to the risk of LBDs (NRC, 2001). Epidemiologic studies demonstrate that trunk posture is an important risk factor for occupational LBDs (Kelsey et al., 1984; Keyserling, 1989; Pope, 1989; Waters et al., 1993). Workers in occupations requiring prolonged static flexion (farm workers, construction, mining, etc.) report unusually high rates of

LBDs (BLS, 1995; Kumar, 2001; NIOSH, 1999). Workers exposed to mild or severe flexion for less than 10% of a work cycle have a LBD risk 4.2 - 4.4 times greater than normal (Marras et al., 1993; Punnett et al., 1991). The odds of a LBD double when workers are exposed to severe flexion for more than 10% of a work cycle (Punnett et al., 1991). Repetitive dynamic flexion has also been shown to be a risk factor for LBDs (Frymoyer et al., 1983; Marras et al., 1993; Punnett & Wegman, 2004). Some have suggested that the biomechanics of injury are related to residual (Cholewicki et al., 1997; Solomonow et al., 1999) or cumulative effects of flexion loading (Kumar, 1990; Norman et al., 1998).

Biomechanical Factors in LBD risk

Spine biomechanics contribute in part to LBD risk. Compressive and shear loads during occupational MMH tasks have been shown to correlate with risk of LBD (Herrin et al., 1986; Karwowski et al., 1994; Kumar, 1990). Psychosocial and personal variables contribute to risk of LBD (Bigos et al., 1986; Videman & Battie, 1999), but evidence suggests high correlations between psychosocial stress and spinal load (Marras et al., 2002) and between spinal load and LBD risk (Norman et al., 1998). Workers in the top 25% of spinal loading exposure suffer 6 times the risk of reported LBD than workers in the bottom quartile of spinal load (Norman et al., 1998). This suggests that occupationally-related LBDs are associated with spine load (Chaffin & Park, 1973). Note that neuromuscular control of lifting and associated muscle forces are the primary contributor to spinal load (Granata & Marras, 1996; Marras & Granata, 1997). Musculoskeletal stability may also be related to LBD risk. Stability describes the ability to maintain equilibrium despite the presence of kinematic and/or control disturbances. The spinal column will buckle (it is unstable) and fail at compressive loads less than 100N if the muscles fail to provide stability (Crisco & Panjabi, 1991). Even minimal loss of stability can momentarily reduce spinal tolerance, thereby allowing injury to occur at compression forces significantly less than NIOSH-recommended guidelines (NIOSH, 1981; Waters et al., 1993). Epidemiologic data suggest that exposure to biomechanically unstable events increases the risk of occupational LBDs (Magora, 1970; Manning et al., 1984; Manning & Shannin, 1981; Omino & Hayashi, 1992; Taimela & Kujala, 1992; Taimela et al., 1993). Thus, biomechanical factors including spinal load and stability likely contribute to the risk of occupationally-related LBDs.

Neuromuscular control influences spinal load and stability. External trunk moment increases with trunk flexion, consequently requiring greater supporting muscle force. Spinal load is influenced by both external and muscle forces so spinal load increases with flexion angle (Chaffin, 1969; Chaffin & Andersson, 1984; Chaffin & Park, 1973; Granata & Marras, 1993, 1995; Schultz & Andersson, 1981). Co-active recruitment of the antagonistic flexor (i.e., abdominal) muscles is also observed during static and dynamic extension exertions (Pope et al., 1987; Zetterberg et al., 1987). This co-contraction can increase compression by 45% and shear load by 70% (Granata & Marras, 1995; Hughes et al., 1995). The spinal load attributed to co-contraction correlates with LBD risk in flexion (Granata & Marras, 1993, 1995), lateral and twisting postures (Marras & Granata, 1995, 1996), and lifting velocity (Granata & Marras, 1993, 1995). This recruitment behavior may be an effort to maintain spinal stability (van Dieën et al., 2003a,b). Co-contraction influences trunk muscle stiffness (Cholewicki et al., 2000; Gardner-Morse & Stokes, 2001) and this plays a critical role in maintaining spinal stability (Cholewicki et al., 1997; Gardner-Morse & Stokes, 1998; Granata & Orishimo, 2001). Therefore, workplace designs that contributes to disturbances of neuromuscular recruitment and/or abnormal active muscle stiffness will influence the control of equilibrium, spinal load and spinal stability (Gardner-Morse et al., 1995; McGill, 2001; McGill & Cholewicki, 2001).

Reflex activity is another important component of neuromuscular control. Reflex response is defined here as transient muscle activity that is observed in response to loading disturbances

(Hodges et al., 1999; Krajcarski et al., 1999; Thomas et al., 1998). Reflexes may contribute to spinal load and stability (Brown et al., 2003; Lavender et al., 1993). Abnormal reflex responses and abnormal muscle recruitment patterns (abnormal co-contraction) have been noted in patients with LBDs (Hodges & Richardson, 1996; Luoto et al., 1996; Marras et al., 2001; Radebold et al., 2001), and Radebold et al. (2000; 2001) highlighted diminished neuromuscular control in low-back patients. However, it is unclear whether this abnormal behavior contributed to the onset of pain or is a compensatory response to the pain (Panjabi, 1992). Reflex as a neuro-mechanical feedback mechanism for stability has been recorded (Granata et al., 2004), but the extent of its contribution to spinal load and stability remains under investigation. One of the few studies in this regard (Marras et al., 1987) suggested that reflexive responses to external disturbances may augment muscle force by 250%. However, that study did not account for low-pass electromechanical filtering effects related to elastic muscle behavior (i.e., stiffness). In other joints of the human body, reflexes may be the primary mechanism of stability (Stokes & Gardner-Morse, 2000). Anything that disturbs normal reflex responses will thus likely influence spinal load and impair the control of spinal stability.

Clearly, diverse neuromuscular factors (co-contraction, muscle stiffness, reflexes) contribute to the control of spinal load and stability. Disturbed neuromuscular control may therefore affect the risk of occupationally-related LBDs. Existing evidence indicates that spinal flexion may temporarily disturb neuromuscular control.

Trunk Flexion and Disturbance of Neuromuscular Control

Flexed postures influence passive support of the spine. Strain in the passive structures of the spine and trunk increases with trunk flexion (Adams & Hutton, 1985; Dolan et al., 1994). At a constant strain (constant spine flexion angle), the load supported by this strain declines with the duration of a flexion task (Solomonow et al., 1999), a process known as “stress relaxation”. At a constant load, the strain (spine flexion) increases with duration of the flexion task (McGill & Brown, 1992), or “creep deformation”. Creep deformation and stress relaxation are analogous biomechanical effects; whether one or the other is recorded depends on the situation or protocol used. Adams and Dolan (1996) observed a 42% reduction in bending moments of cadaveric spines following sustained flexion. Following repeated flexion, the moment was reduced by 17%. These effects have been observed also at low flexion loads (Goel et al., 1988), and recovery from viscoelastic creep deformation occurs in a slow exponential manner (Twomey & Taylor, 1982). Finite element models by Wang et al. (2000) indicated that the history of loading influences passive stiffness (i.e., creep deformation). Similar effects have been observed in animals, wherein cyclic displacements were applied to the L4-L5 supra-lumbar ligament in feline surgical preparations (Solomonow et al., 1999). The force necessary to achieve a 5 mm flexion displacement declined with each repeated cycle (i.e., stress-relaxation). Force recovery was observed when a neutral posture was restored (Gedalia et al., 1999). Similar behaviors were observed with static strain loading (Jackson et al., 2001). Although it has been suggested that these displacements in animal models were excessive, Eversull et al. (2001)[1] concluded that similar behavior occurs in the physiologic range of spine flexion. Thus, spinal flexion in animals causes viscoelastic creep in the passive tissues of the spine.

In humans, similar time-dependent changes in passive spinal laxity has been reported. Passive tissues are believed to support the external moment of the trunk in deep flexion, and indeed paraspinal muscles become myoelectrically silent, a process known as flexion-relaxation (FR) (Kippers & Parker, 1984; Schultz et al., 1985; Toussaint et al., 1995). Note, though, that this load sharing theory for FR does not have unanimous consensus (Olson et al., 2004; Sarti et al., 2001). Under this FR strain, McGill & Brown (1992) observed that the trunk FR angle increased over time in healthy human subjects, and attributed this increase to creep in passive spinal tissues. After returning to an upright posture, the passive laxity slowly (~30 minutes) returned to normal. This spinal creep may be related, at least in part, to intervertebral fluid loss (Adams & Dolan, 1987; Hedman & Fernie, 1995), but nonetheless it causes tissue laxity. Others (Parkinson et al., 2004) have observed similar phenomena as a result of repetitive dynamic lifting, specifically passive stiffness of the trunk decreased following 30 minutes of repeated flexion exertions. As such, it is apparent that flexed postures will cause passive tissue laxity of the spine.

Increased laxity in passive spinal tissues disturbs muscle activity in animals (Klinge et al., 1997). Detailed investigations of the spinal ligaments reveal that they are endowed with sensory receptors (Bogduk et al., 1982, 1998; Cavanaugh et al., 1996; Hirsch et al., 1963; Jackson et al., 1966; Rahlmi et al., 1993). Stimulation of the supraspinous ligament in anesthetized humans during surgeries caused EMG responses in the surrounding muscles, demonstrating a ligamento-muscular reflex loop (Solomonow et al., 1998). Mechanical strain applied to the spinal ligaments in feline models similarly excited myoelectric activity in the surrounding musculature (Kang et al., 2002; Solomonow et al., 1998). Laxity in the passive spinal tissues desensitizes the mechanoreceptors, causing loss of myoelectric response and a potential reduction in stabilizing muscle forces (Solomonow et al., 1999). When the spine was returned to its neutral posture the reflex disturbance slowly returned to normal, with an exponential recovery rate time constant up to 38 minutes (Gedalia et al., 1999). These feline experiments were also performed using static flexion strain, and similar time-dependent decline in EMG response were observed (Jackson et al., 2001). When the spine was returned to its neutral posture, the EMG response demonstrated a slow exponential recovery to normal with a time constant up to several hours. This recovery process following static flexion was preceded by a brief (10-20 minutes) hyper-excitable phase, attributed to possible damage from severe strain imposed by the protocol (Williams et al., 2000). This hyper-excitable phase was not observed with the cyclic flexion protocols. Such neuromuscular disturbances are also observable from flexion strain within the normal physiologic range (Eversull et al., 2001; Solomonow et al., 2001). These animal models indicate that spinal flexion can cause an accumulation of neuromuscular disturbance, with residual effects that influence the control and stability of subsequent exertions. Experiments are necessary, though, to demonstrate this effect in humans.

The rate of accumulation of flexion-induced neuromuscular disturbance in humans is unknown. Changes in passive behavior following static or cyclic flexion have been reported from in vivo human experiments (McGill & Brown, 1992; Parkinson et al., 2004), but few have investigated neuromuscular changes. Two studies (Dickey et al., 2003; Solomonow et al., 2003) examined the lumbar flexion angles at which EMG turns off (FR angle), both before and after periods of trunk flexion in humans. Results from these indicate that the angle of neuromuscular silence is increased (greater flexion) following 10 minutes of static flexion (Solomonow et al., 2003) and following 100 repeated cyclic flexion exertions (Dickey et al., 2003). Studies in animals (Olson et al., 2004; Solomonow et al., 2003) and recent work in humans (Granata et al., 2004) indicate that this passive laxity disturbs neuromuscular behavior following flexion tasks.

Despite the importance of co-contraction in spinal load and stability, few studies have quantified the change in human trunk muscle coactivity following prolonged static flexion tasks and/or repetitive dynamic flexion tasks. Olsen et al. (2004) observed increased paraspinal EMG activity following 9 minutes of cyclic flexion, but it is unclear whether this effect was related to co-contraction, biomechanical changes, or fatigue (slower lift rates were recommended by the authors to avoid fatigue, e.g. 2-4 lifts per minute). Pilot work (Granata et al., 2004) demonstrated significant changes in coactivity following prolonged static flexion. Further work is necessary to quantify the neuromuscular disturbance and neuromuscular recovery rates in order to predict changes in co-contraction, spinal load, and stability associated with work-rest schedules and task design.

Despite the role of active muscle stiffness for control of spinal stability, few studies have quantified the change in human trunk stiffness following flexion tasks. There is evidence for the shoulder (Wilson et al., 1992) and knee (Magnusson et al., 1996) that active muscle stiffness changes as a result of passive stretch, but this effect has not been confirmed in the trunk. Earlier work (Granata et al., 2004) indicated that trunk motion from sudden load is changed following static FR, indicating modified trunk stiffness. Further work is necessary, though, to quantify neuromuscular disturbance and neuromuscular recovery of active (muscle generated) trunk stiffness and stability associated with flexed postures, work-rest schedules, and task design.

Despite the association between paraspinal reflex response, stability, and low-back pain symptoms, few studies have quantified the change in reflexes following static flexion and/or repetitive dynamic flexion tasks in humans. Passive stretch of skeletal muscles, such as might occur in flexed work postures, can reduce muscle spindle excitability thereby inhibiting reflex amplitude (Avela et al., 1999; Rosenbaum & Hennig, 1995). Animal studies and some recent measurements (Granata et al., 2004) demonstrate that static FR influences paraspinal reflex response. Further work is necessary, however, to quantify the role of task design variables on disturbance of reflex, rate of disturbance, and recovery. By augmenting existing trunk biomechanical models, it is possible to investigate how these disturbances influence spinal load and stability.

Research Needs

Although it is recognized that spinal load is increased <u>during</u> work in flexed postures, recent evidence suggests that neuromuscular control of spinal load and stability may also be influenced <u>following</u> exposure to flexed postures. Recovery from these neuromuscular disturbances may be incomplete upon initiation of the subsequent tasks, thereby causing an accumulation of neuromuscular disturbance throughout the work-shift (and possibly beyond). This is analogous to cumulative trauma from the perspective of neuromuscular control. Rate of disturbance growth and the rate of recovery remain to be quantified in humans. The influence of work-task design on these disturbances remains to be quantified. Recent methods and analytical developments permit quantification of changes in active trunk (muscle) stiffness, reflex gain, and co-contraction from in vivo measurements in humans. These data can be used to estimate changes in spinal load and stability resulting from static or cyclic flexion tasks. Therefore, the goal of this application was to quantify the severity and accumulation rate of neuromuscular disturbance from flexed work postures, and to quantify the rate of neuromuscular recovery following working in flexed postures.

References

Adams M.A., Dolan P., and Hutton W.C. Diurnal variations in the stresses on the lumbar spine. Spine 1987;12:130-7.
Adams MA and Dolan P. Time-dependent changes in the lumbar spine's resistance to bending. Clin.Biomech.(Bristol., Avon.) 1996;11:194-200.
Adams M.A. and Hutton W.C. The effect of posture on the lumbar spine. J Bone Joint Surg 1985;67B:625-9.
Andersson G.B.J. Epidemiologic aspects on low back pain in industry. Spine 1981;6:53-60
Andersson G.B.J. The epidemiology of spinal disorders. In: Frymoyer J.W., ed. The Adult Spine: Principles and Practice. New York: Raven Press Ltd., 1981b.
Avela J, Kyrolainen H, and Komi PV. Altered reflex sensitivity after repeated and prolonged

passive muscle stretching. J.Appl.Physiol. 1999;86:1283-91.
Bigos S.J., Battie M.C., Spengler D.D., Fisher L., Nachamsin A., and Wang M.H. Back injuries in industry: A retrospective study. II. Injury factors. Spine 1986;11:1-6.
Brown SH, Haumann ML, and Potvin JR. The responses of leg and trunk muscles to sudden unloading of the hands: implications for balance and spine stability. Clin.Biomech.(Bristol., Avon.) 2003;18:812-20.
(BLS) Bureau of Labor Statistics. Occupational injuries and Illnesses in the U.S. by industry, 1989. U.S.Department of Labor. Bulletin 2379. 1990. Washington D.C. USGPO.
(BLS) Bureau of Labor Statistics. Worker injuries and illnesses by selected characteristics, 1992. U.S.Department of Labor. USDL 94-213. 1994. Washington D.C., USGPO.
(BLS) Bureau of Labor Statistics. Workplace Injuries and Illness. USDL-95-508. 1995. Washington DC, US Dept. of Labor.
(BLS) Bureau of Labor Statistics. Safety and Health Statistics. U.S.Department of Labor. USDL 98-157. 1998. Washington D.C.
Bogduk N, Johnson G, and Spalding D. The morphology and biomechanics of latissimus dorsi. Clin.Biomech.(Bristol., Avon.) 1998;13:377-85.
Bogduk N, Wilson AS, and Tynan W. The human lumbar dorsal rami. J Anat. 1982;134 (Pt 2):383-97.
Brown SH, Haumann ML, and Potvin JR. The responses of leg and trunk muscles to sudden unloading of the hands: implications for balance and spine stability. Clin.Biomech.(Bristol., Avon.) 2003;18:812-20.
Cats-Baril W., Frymoyer J.W. The economics of spinal disorders. In: Frymoyer J.W., Ducker T.B., Hadler N.M., Kostuik J.P., Weinstein J.N., Whitecloud T.S, eds. The Adult Spine. New York: Raven Press, 1991:85-105.
Cavanaugh J.M., Ozaktay C., Yamashita T., and King A.I. Lumbar facet pain: Biomechanics, neuroanatomy and neurophysiology. J.Biomechanics 1996;29:1117-29.
Chaffin D.B. A computerized biomechanical model - Development of and use in studying gross body actions. J.Biomechanics 1969;2:429-41.
Chaffin D.B., Anderson G.B.J. Occupational Biomechanics. New York: John Wiley and Sons, 1984.
Chaffin D.B. and Park K.S. A longitudinal study of low-back pain as associated with weight lifting factors. Am.Ind.Hyg.Ass.J. 1973;34:513-25.
Cholewicki J, Panjabi MM, and Khachatryan A. Stabilizing function of trunk flexor-extensor muscles around a neutral spine posture. Spine 1997;22:2207-12.
Cholewicki J, Simons APD, and Radebold A. Effects of external trunk loads on lumbar spine stability. J.Biomechanics 2000;33:1377-85.
Crisco JJ and Panjabi MM. The intersegmental and multisegmental muscles of the lumbar spine: A biomechanical model comparing lateral stabilizing potential. Spine 1991;16:793-9.
Dickey JP, McNorton S, and Potvin JR. Repeated spinal flexion modulates the flexion-relaxation phenomenon. Clin.Biomech. 2003;18:783-9.
Dolan P, Mannion AF, and Adams MA. Passive tissues help the back muscles to generate extensor moments during lifting. J.Biomechanics; 1994;27:1077-85.
Eversull BSE, Solomonow M, Zhou EEBH, Baratta RV, and Zhu MP. Neuromuscular neutral zones sensitivity to lumbar displacement rate. Clinical Biomechanics 2001;16:102-13.
Frymoyer J.W., Pope M.H., Clements J.H., Wilder D.G., MacPherson B., and Ashikaga T. Risk factors in low back pain: An epidemiologic survey. J.Bone Joint Surg. 1983;65A:213-8.
Gardner-Morse MG and Stokes IAF. The effects of abdominal muscle coactivation on lumbar spine stability. Spine 1998;23:86-92.
Gardner-Morse MG and Stokes IAF. Trunk stiffness increases with steady-state effort. Journal of Biomechanics 2001;34:457-63.

Gardner-Morse MG, Stokes IAF, and Laible JP. Role of muscles in lumbar stability in maximum extension efforts. J.Orthop.Res. 1995;13:802-8.
Gedalia U, Solomonow M, Zhou BH, Baratta RV, Lu Y, and Harris M. Biomechanics of increased exposure to lumbar injury caused by cyclic loading - Part 2. Recovery of reflexive muscular stability with rest. Spine 1999;24:2461-7.
Goel VK, Voo LM, Weinstein JN, Liu YK, Okuma T, and Njus GO. Response of the ligamentous lumbar spine to cyclic bending loads. Spine 1988;13:294-300.
Granata KP and Marras WS. An EMG-assisted model of loads on the lumbar spine during asymmetric trunk extensions. J.Biomechanics 1993;26:1429-38.
Granata KP and Marras WS. The influence of trunk muscle coactivity upon dynamic spinal loads. Spine 1995;20:913-9.
Granata KP, Marras WS. Biomechanical Models in Ergonomics. In: Bhattacharya, McGlothlin, eds. Handbook of Occupational Ergonomics, Theory and Applications. New York: Mercel Dekker, Inc., 1996:115-36
Granata KP and Marras WS. Cost-benefit of muscle co-contraction in protecting against spinal instability. Spine 2000;25:1398-404.
Granata KP and Orishimo K. Response of Trunk Muscle Coactivation to Changes in Spinal Stability. J.Biomechanics 2001;34:1117-23.
Granata KP, Rogers E, and Moorhouse KM. Effects of static flexion-relaxation on paraspinal reflex dynamics. J.Biomech. 2004;24:16-24.
Granata KP, Slota GP, Bennett BE, and Kang HG. Paraspinal muscle reflex dynamics. J.Biomechanics 2004;37:241-7.
Guo H.R., Tanake S., Cameron L.L. et al. Back pain among workers in the United States: National estimates and workers at highest risk. Am.J.Ind.Med. 1995;28:591-602.
Hedman TP and Fernie GR. In vivo measurement of lumbar spinal creep in two seated postures using magnetic resonance imaging. Spine 1995;20:178-83.
Herrin G.A., Jaraiedi M., and Anderson C.K. Prediction of overexertion injuries using biomechanical and psychophysical models. Am.Ind.Hyg.Assoc.J. 1986;47:322-30.
Hirsch C, Ingelmark BE, and Miller M. The anatomical basis for low back pain. Studies on the presence of sensory nerve endings in ligamentous, capsular and intervertebral disc structures in the human lumbar spine. Acta Orthop.Scand. 1963;33:1-17.
Hodges P.W., Cresswell A.G., and Thorensson A. Preparatory trunk motion accompanies rapid upper limb movement. Exp.Brain Res. 1999;124:69-79.
Hodges P.W. and Richardson C.A. Inefficient muscular stabilization of the lumbar spine associated with low back pain. A motor control evaluation of transversus abdominis. Spine 1996;21:2640-50.
Hollbrook T.L., Grazier K., Kelsey J.L, and Stauffer R.N. The frequency of occurence, impact and cost of selected musculoskeletal conditions in the United States. 24-45. 1994. Chicago, Am. Acad. Orthop. Surg.
Hughes R.E., Bean J.C., and Chaffin D.B. Evaluating the effect of co-contraction in optimization models. J.Biomechanics 1995;28:875-8.
Jackson M, Solomonow M, Zhou B., Baratta RV, and Harras M. Multifidus EMG and tension-relaxation recovery after prolonged static lumbar flexion. Spine 2001;26:715-23.
Jackson HC, Winkelmann RK, and Bickel WH. Nerve endings in the human lumbar spinal column and related structures. J Bone Joint Surg Am. 1966;48:1272-81.
Kang YM, Choi WS, and Pickar JG. Electrophysiologic evidence for an intersegmental reflex pathway between lumbar paraspinal tissues. Spine 2002;27:E56-E63.
Karwowski W., Caldwell M., Gaddie P. Relationships between the NIOSH (1991) lifting index, compressive and shear forces on the lumbosacral joint, and low back injury incidence rate based on industrial field study. Proceedings of the Human Factors and Ergonomics Society Annual Meeting 1994:654-657.

Kelsey K.L., Githens P.B., White A.A.III, and et.al. An epidemiologic study of lifting and twisting on the job and risk for acute prolapsed lumbar intervertebral disc. J.Ortho.Res. 1984;2:61-6.
Kelsey J.L. and White A.A.III. Epidemiology and impact on low back pain. Spine 1980;5:133-42.
Keyserling W.M. Analysis of manual lifting tasks: a qualitative alternative to the NIOSH work practices guide. Am.Ind.Hyg.Assoc.J. 1989;50:165-73.
Kippers V and Parker AW. Posture related to myoelectric silence of erectores spinae during trunk flexion. Spine 1984;9:740-5.
Klinge K., Magnusson S.P., Simonsen E.B., Aagard P., Klausen K., and Kjaer M. The effect of strength and flexibility training on skeletal muscle electromyographic activity, stiffness, and viscoelastic stress relaxation response. Am.J.Sport Med. 1997;25:710-6.
Krajcarski S.R., Potvin J.R., and Chiang J. The in vivo dynamic response of the spine to perturbations causing rapid flexion: Effects of pre-load and step input magnitude. Clin.Biomech. 1999;14:54-62.
Kumar S. Cumulative load as a risk factor for back pain. Spine 1990;15:1311-6.
Kumar S. Theories of musculoskeletal injury causation. Ergonomics 2001;44:17-47.
Lavender S.A., Marras W.S., and Miller R.A. The development of response strategies in the preparation for sudden loading to the torso. Spine 1993;18:2097-105.
Luoto S, Taimela S, Hurri A, Aalto H, Pyykko I, and Alaranta H. Psychomotor speed and postural control in chronic low back pain patients. A controlled follow-up study. Spine 1996;21:2621-7.
Magnusson S.P., Simonsen E.B., Aagard P., and Kjaer M. Biomechanical responses to repeated stretches in human hamstring muscle in vivo. Am.J.Sport Med. 1996;24:622-8.
Magora A. Investigation of the relation between low back pain and occupation. Indust.Med. 1970;39:465-71.
Manning D.P., Mitchell R.G., and Blanchfield J.P. Body movements and events contributing to accidental and nonaccidental back injuries. Spine 1984;9:734-9.
Manning D.P. and Shannon H.S. Slipping accidents cause low-back pain in a gearbox factory. Spine 1981;6:70-2.
Marras W.S., Davis K.G., Allread W.G., Maronitis A.B., and Alread. The influence of psychosocial stress, gender, and personality on mechanical loading of the lumbar spine. Spine 2002;25:3045-54.
Marras W.S., Davis K.G., Ferguson S.A., Lucas B.R., and Gupta P. Spine loading characteristics of patients with low back pain compared with asymptomatic individuals. Spine 2001;26:2566-74.
Marras WS, Lavender SA, Leurgans SE, and et.al. The role of dynamic three-dimensional trunk motion in occupationally-related low back disorders: The effects of workplace factors, trunk position and trunk motion characteristics on risk of injury. Spine 1993;18:617-28.
Marras W.S. and Granata K.P. A Biomechanical Assessment and Model of Axial Twisting in the Thoraco-Lumbar Spine. Spine 1995;20:1440-51.
Marras W.S. and Granata K.P. Spine loading during trunk lateral bending motions. J.Biomechanics 1996;30:697-703.
Marras W.S. and Granata K.P. The development of an EMG-assisted model to assess spine loading during whole-body free-dynamic lifting. J.Electromyo.Kinesiol. 1997;7:259-68.
Marras W.S., Rangarajulu S.L., and Lavender S.A. Trunk loading and expectation. Ergonomics 1987;30:551-62.
McGill S.M. Low back stability: From formal description to issues for performance and rehabilitation. Exer.Sport Sci.Rev. 2001;29:26-31.
McGill SM and Brown S. Creep response of the lumbar spine to prolonged full flexion. Clin Biomech. 1992;7:43-6.
McGill SM and Cholewicki J. Biomechanical basis for stability: An explanation to enhance clinical utility. J.Ortho.Sports Phys.Ther. 2001;31:96-100.

Moorehouse KM, Granata K P. Trunk stiffness and dynamics during active extension exertions. J.Biomechanics 2005; 38:2000-7.
NIOSH. A Work Practices Guide for Manual Lifting. 81-122. 1981. Cincinnati, Oh, U.S. Dept. of Health and Human Services (NIOSH).
(NIOSH) National Institute for Occupational Safety and Health. Survey of Occupational Injuries and Illnesses. 1999. Washington DC.
(NRC) National Research Council. Musculoskeletal Disorders and the Workplace. Washington DC: National Academy Press, 2001.
(NSC) National Safety Council. Accident Facts. 1989. Chicago, IL.
Norman RW, Wells R, Neumann P et al. A comparison of peak vs cumulative physical work exposure risk factors for the reporting of low back pain in the automotive industry. Clin.Biomech. 1998;13:561-73.
Olson MW, Li L, and Solomonow M. Flexion-relaxation response to cyclic lumbar flexion. Clin.Biomech.(Bristol., Avon.) 2004;19:769-76.
Omino K and Hayashi Y. Preparation of dynamic posture and occurrence of low back pain. Ergonomics 1992;35:693-707.
Panjabi M.M. The stabilizing system of the spine. Part I Function, dysfunction, adaptation and enhancement. J.Spinal Disorders 1992;5:383-9.
Parkinson RJ, Beach TA, and Callaghan JP. The time-varying response of the in vivo lumbar spine to dynamic repetitive flexion. Clin.Biomech.(Bristol., Avon.) 2004;19:330-6.
Pope M.H. Risk indicators in low back pain. Ann.Med. 1989;21:387-92.
Pope M.H., Svensson M.S., Andersson G.B.J., Broman H., and Zetterberg C. The role of prerotation of the trunk in axial twisting efforts. Spine 1987;12:1041-5.
Praemer A., Furner S., and Rice D.P. Musculoskeletal conditions in the United States. 23-33. 1992. Park Ridge, IL., Am. Acad. Orthop. Surg.
Punnett L, Fine LJ, Keyserling WM, Herrin GD, and Chaffin DB. Back disorders and non-neutral trunk postures of automobile assembly workers. Scand.J.Work Envir.Health. 1991;17:337-46.
Punnett L and Wegman DH. Work-related musculoskeletal disorders: the epidemiologic evidence and the debate. J Electromyogr.Kinesiol. 2004;14:13-23.
Radebold A, Cholewicki J, Panjabi MM, and Patel TC. Muscle response pattern to sudden trunk loading in healthy individuals and in patients with chronic low back pain. Spine 2000;25:947-54.
Radebold A, Cholewicki J, Polzhofer GA, and Green TP. Impaired postural control of the lumbar spine is associated with delayed muscle response times in patients with chronic idiopathic low back pain. Spine 2001;26:724-30.
Rhalmi S, Yahia LH, Newman N, and Isler M. Immunohistochemical study of nerves in lumbar spine ligaments. Spine 1993;18:264-7.
Rosenbaum D and Hennig EM. The influence of stretching and warm-up exercises on Achilles tendon reflex activity. J.Sports Sci. 1995;13:481-90.
Rosenstock L, Olenec C., Wagner G.R. Public health policy forum. The National Occupational Research Agenda: A model of broad stakeholder input into priority setting. Am J.Pub.Health 1998;88:353-6.
Rowe M.L. Low back disability in industry: An updated position. J.Occ.Med. 1971;13:476-8.
Sarti MA, Lison JF, Monfort M, and Fuster MA. Response of the flexion-relaxation phenomenon relative to the lumbar motion to load and speed. Spine 2001;26:E421-E426.
Schultz AB and Andersson GB. Analysis of loads on the lumbar spine. Spine 1981;6:76-82.
Schultz AB, Haderspeck-Grib K, Sinkora G, and Warwick DN. Quantitative studies of the flexion-relaxation phenomenon in the back muscles. J Orthop.Res. 1985;3:189-97.

Solomonow M, Baratta RV, Banks A, Freudenberger C, and Zhou BH. Flexion-relaxation response to static lumbar flexion in males and females. Clin.Biomech.(Bristol., Avon.) 2003;18:273-9.
Solomonow M, Eversull E, He ZB, Baratta RV, and Zhu MP. Neuromuscular neutral zones associated with viscoelastic hysteresis during cyclic lumbar flexion. Spine 2001;26:E314-E324.
Solomonow M, Zhou B, Baratta RV, Lu Y, and Harras M. Biomechanics of increased exposure to lumbar injury caused by cyclic loading: 1. loss of reflexive muscular stabilization. Spine 1999;24:2426-34.
Solomonow M, Zhou B, Harras M, Lu Y, and Baratta RV. The ligamento-muscular stabilizing system of the spine. Spine 1998;23:2552-62.
Spengler D.M., Bigos S.J., Martin N.A., Zeh J., Fisher L., and Nachemson A. Back injuries in industry: A retrospective study. I. Overview and cost. Spine 1986;11:241-5.
Stokes I.A.F. and Gardner-Morse M.G. Strategies used to stabilize the elbow joint challenged by inverted pendulum loading. J.Biomechanics 2000;33:737-43.
Taimela S. and Kujala U.M. Reaction times with reference to musculoskeletal complaints in adolescence. Perceptual & Motor Skills 1992;75:1075-82.
Taimela S., Osterman K., Alaranta H., Soukka A., and Kujala U.M. Long psychomotor reaction time in patients with chronic low-back pain: preliminary report. Arch.Phys.Med.Rehab. 1993;74:1161-4.
Thomas J.S., Lavender S.A., Corcos D.M., and Andersson G.B.J. Trunk kinematics and trunk muscle activity during a rapidly applied load. J.Electromyo.Kinesiol. 1998;8:215-25.
Toussaint H.M., Winter A.F., Haas Y., Looze M.P., Van Dieen J.H., and Kingma I. Flexion relaxation during lifting : Implications for torque production by muscle activity and tissue strain at the lumbo- sacral joint. J.Biomechanics 1995;28:199-210.
Twomey LT and Taylor JR. Flexion creep deformation and hysteresis in the lumbar vertebral column. Spine 1982;7:116-22.
van Dieën JH, Cholewicki J, and Radebold A. Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine. Spine 2003a;28:834-41.
van Dieën JH, Kingma I, and van der Burg JC. Evidence for a role of antagonistic cocontraction in controlling trunk stiffness during lifting. J.Biomech. 2003b;36:1829-36.
Videman T. and Battie M.C. Spine update - The influence of occupation on lumbar degeneration. Spine 1999;24:1164-8.
Wang JL, Parnianpour M, Shirazi-Adl A, Engin AE. Viscoelastic finite-element analysis of a lumbar motion segment in combined compression and sagittal flexion. Effect of loading rate. Spine 2000;25:310-8.
Waters T.R., Baron S.L., Piacitelli L.A. et al. Evaluation of revised NIOSH lifting equation. A cross-sectional epidemiologic study. Spine 1999;24:386-95.
Waters TR, Putz-Anderson V, Garg A, and Fine LJ. Revised NIOSH equation for the design and evaluation of manual lifting tasks. Ergonomics 1993;36:749-76.
Williams M, Solomonow M, Zhou BH, Baratta RV, and Harris M. Multifidus spasms elicited by prolonged lumbar flexion. Spine 2000;25:2916-24.
Wilson G.J., Elliott B.C., and Wood G.A. Stretch shorten cycle performance enhancement through flexibility training. Med.Sci.Sport Exerc. 1992;24:116-23.
Zetterberg C, Andersson GB, and Schultz AB. The activity of individual trunk muscles during heavy physical loading. Spine 1987;12:1035-40.

2. Specific Aims

Four experiments, addressing four specific aims, were proposed and completed. As a set, these were designed to address the effects of prolonged and/or repetitive trunk flexion on: neuromuscular disturbances, neuromuscular recovery, and the accumulation of neuromuscular disturbances. Here, neuromuscular disturbances refer to changes in intrinsic mechanical aspects of the spine (i.e., stiffness) and also reflexive responses of the paraspinal muscles.

Specific Aim #1:
Quantify the severity of neuromuscular disturbance associated with the magnitude and duration of external loads during full flexion in humans (creep-induced effects).

Specific Aim #2:
Quantify the severity of neuromuscular disturbance associated with the magnitude and duration of static lumbar flexion in humans (load-relaxation effects).

Specific Aim #3:
Quantify the accumulation of neuromuscular disturbance associated with repeated static lumbar flexion in humans, and specifically the modifying effects of flexion duration and duty (work-rest) cycle (creep-induced effects).

Specific Aim #4:
Quantify the accumulation of neuromuscular disturbance associated with repetitive lifting in humans, and specifically the modifying effects of flexion angle and lifting rate (load-relaxation effects).

3. Aim 1: Influences of Duration and External Load on Creep-Induced Effects on Neuromuscular Behaviors

Bazrgari B, Hendershot B, Muslim K, Toosizadeh N, Nussbaum MA, Madigan ML: [2011] Disturbance and recovery of trunk mechanical and neuromuscular behaviors following prolonged trunk flexion: influences of duration and external load on creep-induced effects. Ergonomics 54:1043-1052.

Abstract
Trunk flexion results in adverse mechanical effects on the spine and is associated with a higher incidence of low back pain. To examine the effects of creep deformation on trunk behaviors, participants were exposed to full trunk flexion in several combinations of exposure duration and external load. Trunk mechanical and neuromuscular behaviors were obtained pre- and post-exposure and during recovery using sudden perturbations. Intrinsic trunk stiffness decreased with increasing flexion duration and in the presence of the external load. Recovery of intrinsic stiffness required more time than the exposure duration and was influenced by exposure duration. Reflexive trunk responses increased immediately following exposure but recovered quickly (~2.5 min). Alterations in reflexive trunk behavior following creep deformation exposures may not provide adequate compensation to allow for complete recovery of concurrent reductions in intrinsic stiffness, which may increase the risk of injury due to spinal instability. Practioner Summary: An increased risk of low back injury may result from flexion-induced disturbances to trunk behaviors. Such effects, however, appear to depend on the type of flexion exposure, and have implications for the design of work involving trunk flexion.

Introduction
Low back pain (LBP) is the most important work-related musculoskeletal disorder, and continues to have a high prevalence and substantial economic burden (Baldwin 2004, Luo et al. 2004, Katz 2006, Dagenais et al. 2008, Manchikanti et al. 2009). Epidemiological studies have identified several LBP risk factors, in particular prolonged or repetitive torso flexion (Hoogendoorn et al., 2000), though some controversy remains regarding causality (Wai et al. 2010, Kuijer et al. 2011, McGill 2011). Nonetheless, flexed working postures are frequent in mining (Gallagher 2008), construction (Boschman et al. 2011), and in agricultural work (Fathallah et al. 2008, Fathallah 2010), occupations that all have high LBP incidence rates (BLS 2009). With forward trunk flexion from the upright standing posture, there is a corresponding increase in external moments on the lumbar spine, increasing the requirement for force development in the posterior musculature. Moment arms of these muscles, however, decrease with trunk flexion (Macintosh et al. 1993, Jorgensen et al. 2003), thereby requiring larger muscle forces for a given external demand and potentially leading to elevated spinal loads in flexed postures (Arjmand et al. 2006, Bazrgari et al. 2007). Flexed trunk postures may also compromise neuromuscular control of spinal curvature as a consequence of decreased trunk proprioception, leading to spinal instability (Wilson and Granata 2003, Gade and Wilson 2007).

In addition to these adverse mechanical effects of trunk flexion, both active (neuromuscular) and passive (mechanical) trunk tissue behaviors can be influenced if flexed postures occur frequently and/or for prolonged durations. Creep deformation of passive trunk tissues occurs during prolonged flexed postures, manifesting in an increased trunk flexion angle (Twomey and Taylor 1982, McGill and Brown 1992) and an increased activation of the active neuromuscular system (Dickey et al. 2003, Solomonow et al. 2003b, Shin and Mirka 2007). Alternatively, stress relaxation of passive tissues occurs with sustained flexion and is followed by a substantial decrease in passive spine stiffness (Adams and Dolan 1996) and an angle-dependent decrease in whole-trunk flexural resistance (Hendershot et al. 2011). Furthermore, passive stretching of skeletal muscles reduces their active force-generating capacity (Fowles et al. 2000, Weir et al. 2005), diminishes muscle spindle excitability (Avela et al. 1999), and may alter the excitability of the ligament-muscle reflex loop (Le et al. 2009, Solomonow 2009).

The effects of trunk flexion on passive tissues may compromise spinal stability, which would require increased contributions from the active neuromuscular system. Yet, associated changes in muscle force-generating capacity, muscle spindle excitability, and sensitivity of ligament-muscle reflex loop diminish the efficiency of the neuromuscular system in generating the appropriate responses. Collectively, these changes may adversely affect mechanics of the spinal column and increase injury risk due to excessive spinal loads and/or spinal instability (Panjabi 1992a, 1992b). As such, quantifying the acute changes to active neuromuscular and passive mechanical trunk behaviors and the resultant effects on spine biomechanics (i.e., spinal loads and stability) following prolonged flexed posture is important for better understanding of LBP etiology, and will help improve work design and/or work-rest cycles in occupations involving frequent and/or prolonged flexed postures.

Due to the diversity of occupational exposures, workers may experience different levels of creep deformation and/or stress relaxation, thus requiring separate quantification of the effects of creep deformation and stress relaxation on trunk behaviors. Recently, we have developed a sudden perturbation paradigm to obtain measures related to trunk behaviors, and the effects of flexion angle and duration on active and passive trunk behaviors following a stress relaxation experiment were reported earlier (Hendershot et al. 2011). In continuation, the objective of the present work was to investigate the effects of flexion duration and external load on active and passive trunk behaviors following creep deformation. It was hypothesized that: 1) the severity of changes in trunk behavior increase with flexion duration and external load, and 2) recovery is prolonged and contingent on the severity of immediate changes. Moreover, and related to the potential causal role of flexed postures for LBP, we hypothesized that 3) viscoelastic changes in passive trunk tissue following prolonged flexed posture are not adequately compensated by the active neuromuscular system.

Methods

Participants

Twelve young adults participated after completing informed consent procedures approved by the Virginia Tech Institutional Review Board. None had any self-reported history of low-back pain or current medical conditions. Participants included six males with mean (SD) age, stature, and body mass of 23 (3) yr, 181.3 (7.9) cm, and 71.3 (7.3) kg, respectively; corresponding values for the six females were 24 (3) yr, 166 (6.1) cm, and 60.2 (2.2) kg. A relatively young group of participants was included to avoid potential influences related to age.

Experimental Design and Procedures

Creep deformation in the lumbar spine was induced by full trunk flexion in six conditions involving all combinations of three exposure durations (2, 4, and 10 min) and two external loads (none and 29 N). The specific levels of duration and load were intended to represent a range of potential occupational exposures with and without handling a small object (e.g., tool or material). A repeated-measures design was used, in which each participant experienced each condition in a counterbalanced order (one 6x6 Latin Square for each gender) and on different days separated by at least 72 hours.

Experimental trials imposed full, relaxed flexion of the trunk to induce creep deformation. Participants stood in a rigid metal frame and adjustable straps were used to restrain the pelvis and lower limbs. Subsequently, they slowly (~3 sec) flexed forward from an upright standing and remained in full flexion with minimal trunk muscle activity for the designated duration with their head facing down and their arms relaxed and hanging down (Fig. 1). For conditions involving a load, two 14.5 N weights were attached to participants' wrists. Following the flexion exposure, participants returned to and maintained an upright posture with their head facing forward (Fig. 1), while still strapped to the frame, for 60 min to assess post-exposure recovery. Recovery measures were obtained 2.5, 5, 10, 20, 30, 40, 50, and 60 min after exposure. The typical delay between the end of a flexion exposure and the first post-exposure measurement was ~15 sec.

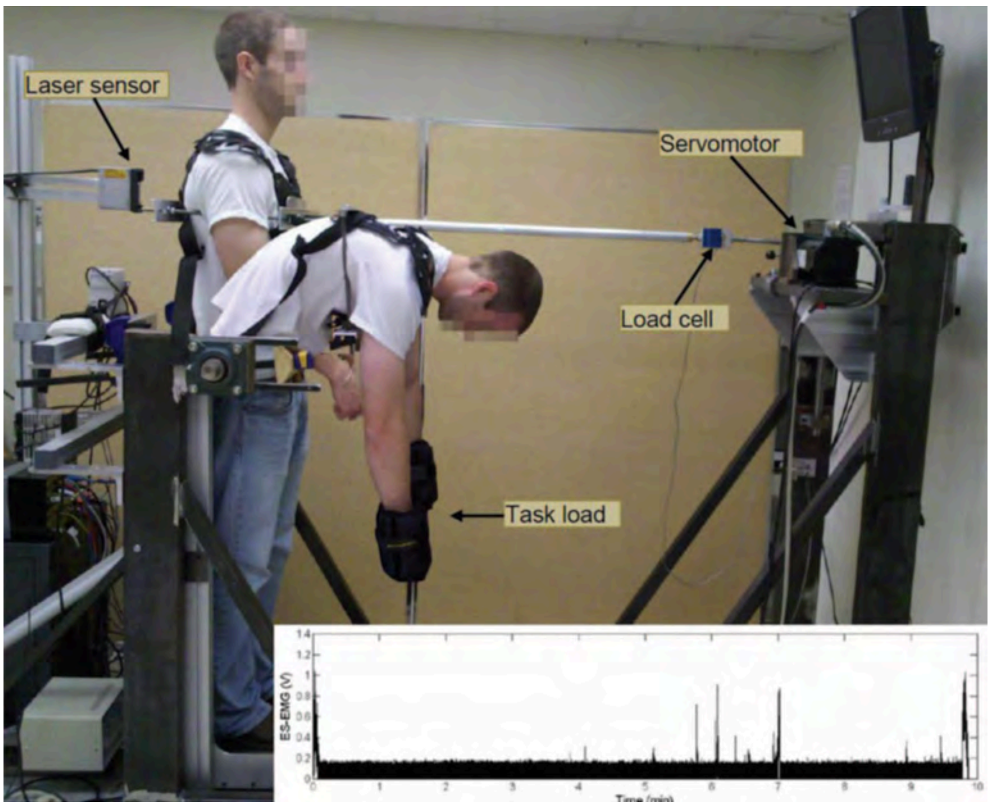

Figure 1: Experimental situation, demonstrating a participant in the sudden perturbation setup superimposed with a picture of the same participant in a flexed posture with external load. A sample of raw erector spinae (L3) EMG, obtained during a 10-minute flexion exposure, demonstrates EMG spikes during the creep period. Note that the initial and final EMG activities are related to the initial flexion and final extension phases of exposure.

Participants were instrumented with a 6 DOF inertial measurement unit (IMU: Xsens Technologies XM-B-XB3, Enschede, Netherlands) placed over the T10 vertebral process to measure trunk flexion, and bipolar Ag/AgCl surface electrodes to measure electromyographic (EMG) activity of select bilateral trunk muscles (i.e., erector spinae muscle at the L1 and L3 level, rectus abdominus, and external oblique). Raw EMG signals were pre-amplified (x100) near the collection site, bandpass filtered (10-500Hz), amplified, and converted to RMS in hardware (Measurement Systems Inc., Ann Arbor, MI, USA). Kinematic data were sampled at 100 Hz and EMG at 1000 Hz.

Pre- and post-exposure measures of trunk behaviors were obtained using a sudden-perturbation paradigm identical to that described in our earlier work (Hendershot et al. 2011). Briefly, this involved application of a pseudorandom sequence of twelve ±5mm anterior-posterior perturbations to the trunk (at ~T8) via a servomotor (Kollmorgen, Radford, VA), rigid rod, and chest harness (Fig. 1). The total length of perturbation sequences was ~ 45 s and each perturbation was completed within 40 ms, which is less than typical erector spinae reflex delays. Pseudorandom delays between each perturbation were used to prevent anticipation of perturbation timing by the participants and hence reduce potential confounding from variations in anticipatory muscle activation. Postural displacements were measured with a high-speed, high-accuracy CCD laser displacement sensor (Keyence, Osaka, Japan) and the motor encoder, while applied forces were measured using an in-line load cell (Interface SM2000, Scottsdale, AZ, USA). For ~3 sec prior to and during the perturbation sequences, participants maintained a constant submaximal extensor effort (or "preload"). The target effort was set to 10% of maximum voluntary RMS EMG in the bilateral L3 erector spinae, and which was determined at the beginning of each experimental session. During perturbations, real-time visual feedback of the target effort was provided. Mean (SD) baseline preloads for the submaximal efforts were 62.1 (19.6) N for males and 57 (12.4) N for females.

Outcome measures and data analysis

Creep deformation throughout each exposure period was characterized by changes in trunk flexion measured by the IMU. For each anteriorly-directed perturbation, the latent period was determined as the time between perturbation onset and reflexive muscle response (Zhang et al. 1999, Granata et al. 2004); the former was determined when the absolute value of measured trunk velocity (from laser) exceeded zero, and the latter identified when erector spinae reflex response peaks exceeded two standard deviations above mean activity prior to the perturbations (Hendershot et al. 2011). Trunk mechanical behaviors during the latent period (i.e., intrinsic properties) were identified by relating measured trunk kinematics to trunk kinetics (both measured in horizontal direction at T8), and by modeling the trunk as a single degree-of-freedom mass-spring-damper system. An extra mass-spring-damper element was included in the model to account for mechanical properties of connecting elements between the motor and spine, specifically the connecting rod, harness, and soft tissues such and padding at the trunk-harness interface (Hendershot et al. 2011, Bazrgari et al. 2011). In this analysis, trunk damping was forced to zero (see Hendershot et al. 2011 for detailed discussion) so that any alterations in intrinsic trunk behavior could be represented by changes in trunk stiffness; earlier work also suggested that trunk damping may be negligible (Cholewicki et al. 2000). Model parameters (apparent mass, stiffness, and damping) were estimated using a least-squares curve fit in MATLAB™ (MathWorks, Natick, MA, USA). To characterize trunk reflexive behavior, reflex forces were first estimated by subtracting the model-estimated intrinsic force contribution from the total measured trunk response (i.e., trunk reaction force measured by the inline load cell). Magnitude and timing (with respect to perturbation onset) of the maximum reflex force were quantified to represent the overall trunk reflexive behavior; this analysis was limited to a time window of 150 ms following reflex onset to avoid voluntary responses. The instantaneous reflex force during this same time window was also correlated to time-shifted (equal to reflex delay) trunk velocity to estimate reflex gain (Moorhouse and Granata 2007).

Pre-exposure differences in trunk behaviors between genders were evaluated using unpaired *t*-tests. Post-exposure measures were normalized to pre-exposure values [(post-pre)/pre] and acute effects of flexion duration, load, and gender were assessed using mixed-factor analyses of variance (ANOVA). No significant deviations from parametric model assumptions were evident. A repeated-measures MANOVA was used to assess the effects of these same factors over the recovery period (one-hour post-exposure); where sphericity violations were found, the

Geisser-Greenhouse correction was used. When relevant, post-hoc pairwise comparisons were performed using Tukey's HSD. Data from one trial of a male participant (2 min, no load) and one female participant (4 min, with load) were excluded due to measurement errors. Summary results are presented as means (SD). All analyses were done using JMP™ (Version 8, SAS Institute Inc., Cary, NC), and statistical significance was concluded when $p<0.05$.

Results

Pre-exposure

Estimated apparent trunk mass was larger among males than females (Table 1), and was linearly correlated with whole-body mass ($R^2=0.8$). Intrinsic trunk stiffness was similarly higher among males. Muscle reflex delays were comparable between genders, though females demonstrated a significantly larger reflex gain and force magnitude. Initial flexion angles were significantly larger among females than males, with respective values of 80 (19) and 72 (13) degrees. Holding the external load increased initial flexion angles by < 1 deg ($t_{(65)} = 0.21$, $p=0.83$).

Table 1. Pre-exposure measures of trunk behaviors. Mean (SD) values are shown for each gender, and significant differences between genders are indicated by bolded *p*-values.

Measure	Males	Females	*t*	*p*-value
Apparent Mass (kg)	20.3 (3.0)	18.6 (2.3)	$t_{(63.5)} = 2.7$	**0.009**
Intrinsic Stiffness (N/m)	8418 (1201)	5738 (908)	$t_{(63.1)} = 10.4$	**<0.0001**
Reflex Delay (ms)	60.1 (3.4)	60.8 (3.4)	$t_{(67)} = -0.9$	0.37
Reflex Gain (Ns/m)	1085 (278)	1315 (219)	$t_{(64.5)} = -3.84$	**0.0003**
Max. Reflex Force (N)	169 (34)	218 (24)	$t_{(61.7)} = -6.93$	**<0.0001**
Timing of Max. Reflex Force (ms)	158.2 (8.7)	151.8 (6.2)	$t_{(61.3)} = 3.54$	**0.0008**
Initial Flexion Angle (deg)	72 (13)	80 (19)	$t_{(59)} = -2.07$	**0.043**

During exposure

Creep deformation during flexion exposures across all conditions was 8.1 (4.6) degrees. These were not different ($F_{(1,7)}=0.36$, $p=0.57$) between females (8.3 (5.9) degrees) and males (7.6 (3.2) degrees). Creep deformation increased with increasing exposure duration ($F_{(2,39)} = 3.87$; $p=0.03$), with values of 5.9 (3.6), 8.6 (4.5), 9.4 (5.2) degrees following 2, 4, and 10 minutes of exposure, respectively. External load did not affect creep ($F_{(1,37)} = 0.01$; $p=0.91$), and the overall difference between loaded and unloaded conditions was ~0.1 deg. During flexion exposures, discrete EMG spikes were observed in 76% and 34% of trials among males and females, respectively (representative example given in Fig. 1).

Immediate post-exposure behavior

Apparent trunk mass decreased following exposure, but was not affected by duration, load, or gender (Table 2). Intrinsic stiffness decreased as exposure duration increased and decreased as the external load increased (Fig. 2 and Table 2). Intrinsic stiffness decreased by 4.8 (3.9), 4.7 (4.3), and 9.3 (4.7) % following 2, 4, and 10 minutes of prolonged full-flexed posture and by 7.8 (4.7), and 4.8 (4.5) % with and without external load, respectively. Reflex delays were unaffected by flexion exposure, however, reflex gains as well as magnitudes and timing of the maximum reflex forces significantly increased across all conditions, by 79.5 (48.1) Ns/m, 17.7 (6.5) N, and 3.9 (4.7) ms, respectively (Fig. 2 and Table 2). Reflex gains were affected by flexion duration, with increases of 3.4 (7.1), 8.3 (7.7) and 10.6 (6) % after 2, 4, and 10 minutes, respectively. Maximum reflex forces increased similarly with flexion duration by 6.8 (8.2), 10.2 (7.8) and 12.1 (5.7) % after 2, 4, and 10 min exposures, respectively. Muscle activity (total across all muscles) during the ~3 seconds prior to perturbations was comparable between all conditions and genders (p>0.47), with respective pre- and post-exposure values of 0.043 (0.009) and 0.044 (0.011) mV.

Table 2. Immediate effects of trunk flexion exposure on trunk behaviors (statistical significance indicated by shaded cells). The "Overall" column indicates paired comparisons (post vs. pre-exposure) across all conditions, and main effects are results from ANOVA (no interaction effects were significant).

Variable	Overall	Main effect		
		Duration	Load	Gender
Apparent Mass	$t_{(69)}$ = -3.79, p=0.0003	$F_{(2,44)}$ = 0.3, p=0.75	$F_{(1,44)}$ = 0.11, p=0.74	$F_{(1,10)}$ = 0.69, p=0.42
Intrinsic Stiffness	$t_{(68)}$ = -9.92; p<0.0001	$F_{(2.43)}$ = 9.9, p=0.0003	$F_{(1.43)}$=11.5, p=0.001	$F_{(1,10)}$ =3.87, p=0.07
Reflex delay	$t_{(69)}$ = 0.98, p=0.33	$F_{(2,42)}$ = 1.56; p=0.22	$F_{(1,42)}$ = 0.05; p=0.83	$F_{(1,9)}$ = 1.2, p=0.31
Reflex Gain	$t_{(69)}$ = 8.34; p<0.0001	$F_{(2,43)}$= 9.3, p=0.0004	$F_{(1,43)}$ = 2.3, p=0.14	$F_{(1,10)}$ =0.7, p=0.41
Max. Reflex Force	$t_{(69)}$ = 11.2; p<0001	$F_{(2,43)}$=3.7, p=0.033	$F_{(1,43)}$ = 3.1 p=0.086	$F_{(1,10)}$ = 1.8, p=0.21
Timing of Max. Reflex Force	$t_{(67)}$ = 4.79; p<0.0001	$F_{(2,44)}$ = 0.17, p=0.85	$F_{(1,44)}$ = 0.04, p=0.84	$F_{(1,11)}$ = 1.7, p=0.21

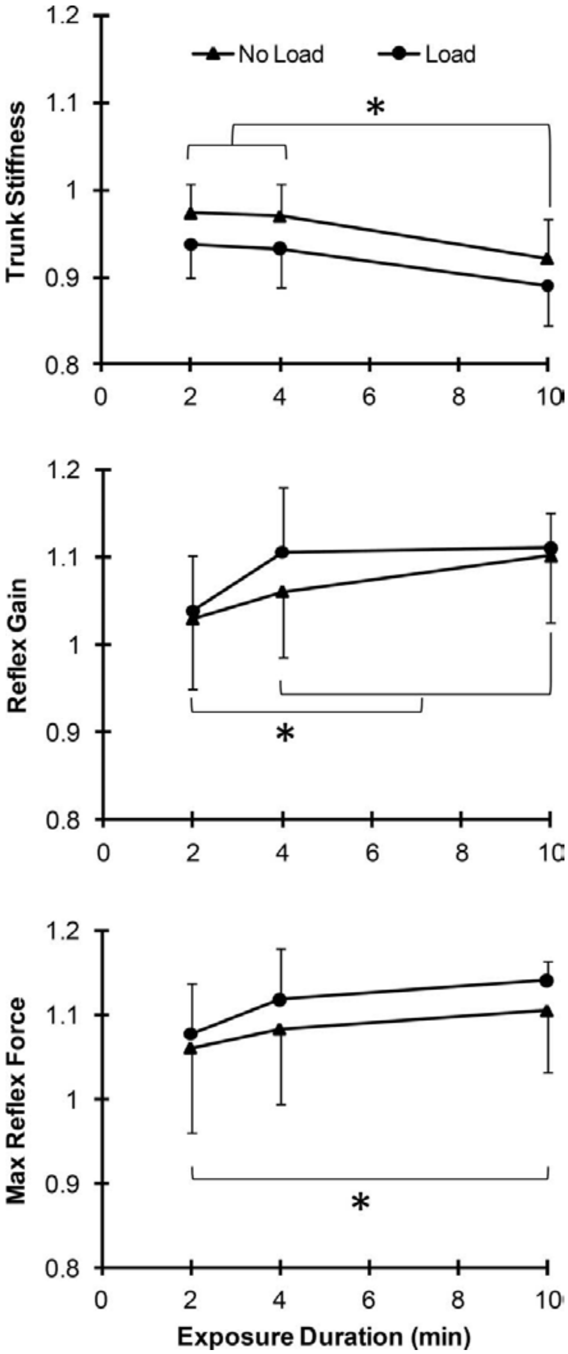

Figure 2: Effects of flexion duration and external load on normalized intrinsic trunk stiffness, reflex gain, and the maximum reflex force. Error bars indicate standard deviations, and * indicates post-hoc groupings with respect to duration.

Recovery behavior

Apparent trunk mass and reflex delays remained unchanged throughout the recovery period (p = 0.44 and p = 0.76, respectively). Recovery of intrinsic trunk stiffness was unaffected by external load (p=0.49) or gender (p=0.53), but differed between exposure durations (p=0.0006). Using the criteria of a non-significant difference from pre-exposure values, the time required to recover intrinsic stiffness was directly related to the exposure duration; specifically, 2.5, 20, and

50 minutes were required following 2, 4, and 10 minutes of flexion exposure (Fig. 3). Recovery of reflex gains, maximum reflex forces, and the timing of maximum reflex in post-exposure perturbation trials were consistent across the three exposure durations (p=0.31, p=0.32, and p=0.77) and two external loads (p=0.39, p=0.46, and p=0.74). Post-exposure changes in the reflex gains and maximum reflex forces were only significant for ~ 2.5 minutes following exposure (Fig. 4), indicating a fast recovery of reflex behavior as opposed to intrinsic stiffness. Recovery of reflex behaviors, however, differed significantly between genders. Males exhibited consistently larger reflex gains (p=0.022), larger maximum reflex forces (p=0.0007), and longer times to maximum reflex force (p=0.0001) during recovery from the longest (10 min) flexion exposure (Fig. 4). Total muscle activities prior to perturbations remained constant throughout the recovery period and were not affected by condition or gender (p>0.32).

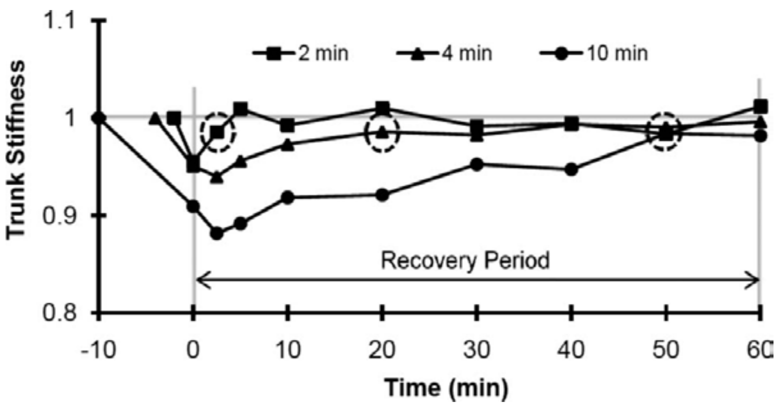

Figure 3: Recovery of normalized intrinsic trunk stiffness following exposure to 2, 4, and 10 minutes of flexion. Times at which trunk stiffness recovered (i.e., non-significant difference between post- and pre-exposure values) are depicted by the circled data points. Time = 0 indicates the end of the exposure period.

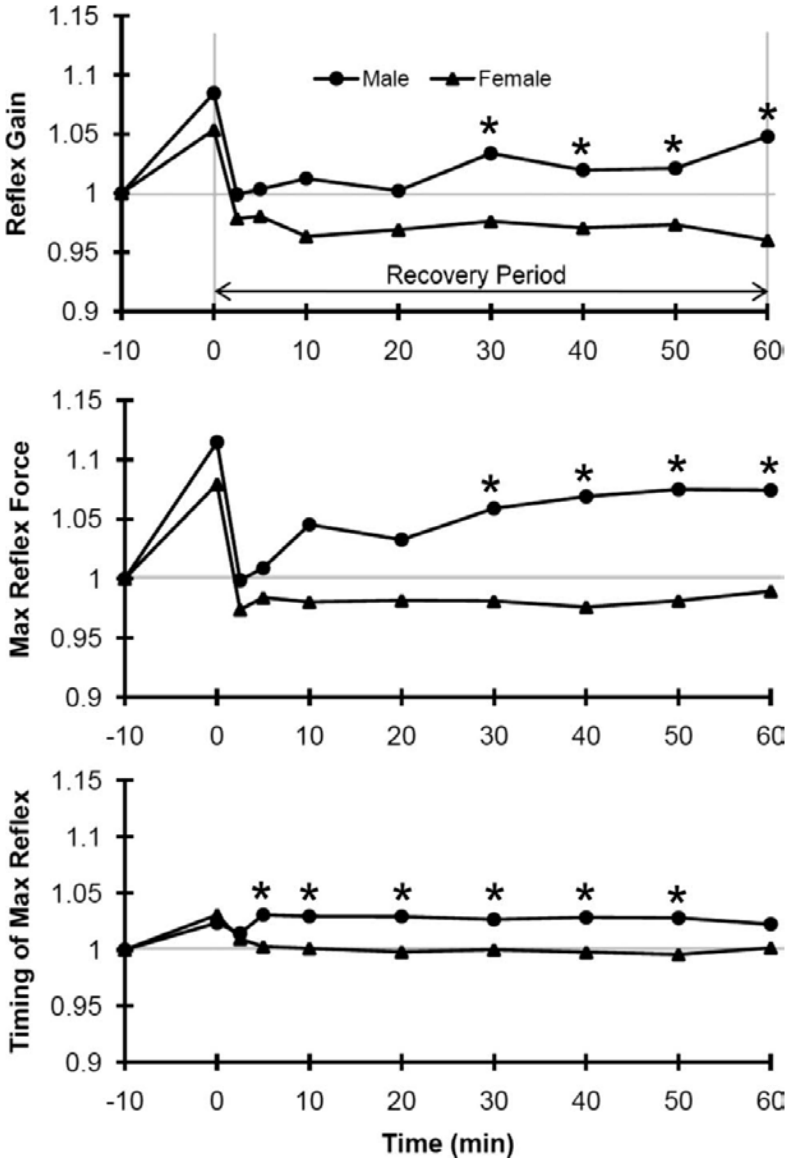

Figure 4: Gender differences in the disturbance and recovery patterns of normalized reflex gain, magnitude, and timing of maximum reflex force following the 10-minute flexion exposure. Time = 0 indicates the end of the exposure period and * denotes a significant difference between genders.

Discussion

Creep deformation

Observed creep following 10 minutes of flexion (mean = 9.4 deg) was larger than earlier values of ~4 deg based on lumbar flexion angle (McGill and Brown 1992, Shin and Mirka 2007). Owing to difficulties with attaching an IMU unit on the sacrum in the present study, creep deformation was assessed from trunk rather than lumbar flexion. Though participants' pelvises were restrained in the present study, a portion of the recorded deformation was likely due to creep deformation of lower body parts that were translated into trunk rotation via pelvic rotation. Reports of hamstring soreness by our participants support this idea, as a complete restraining of the pelvis would likely unload the lower limbs and should not lead to such soreness. The difference between our findings and those of earlier studies is also consistent with differences in creep deformation in a seated flexed posture based on trunk vs. lumbar flexion angles (Solomonow et al. 2003a).

A mean increase of ~ 1 degree in initial trunk flexion angle was found with 29 N of external load, consistent with a ~1.5-3 degree increase in trunk flexion relaxation angle reported earlier with ~ 100 N external loads (Kippers and Parker 1984, Gupta 2001, Dickey et al. 2003). Dickey et al. (2003) have also suggested that the lumbar spine may not experience any additional creep with an external load, again consistent with the current results. It appears that trunk mass times some minimum duration (in the present study) is sufficient to bring the trunk viscoelastic tissues to their plateau region of creep deformation. EMG spikes, observed during flexion exposures (cf. Fig 1), have been reported earlier (Solomonow et al. 2003a) and been suggested to result from micro-damage to ligamentous tissue under sustained deviated postures (Solomonow et al. 2003a).

Intrinsic trunk behavior

Detailed discussions on the methods used to estimate trunk intrinsic properties can be found in our earlier work (Bazrgari et al 2011). Consistent with our earlier studies (Miller et al. 2010, Hendershot et al. 2011), females demonstrated a smaller intrinsic response (smaller apparent mass and intrinsic stiffness) to sudden perturbations. Since trunk apparent mass was found to be highly correlated with whole-body mass, the prediction of smaller trunk apparent mass for females in the present study can be related to a lower whole-body mass (i.e., 71.3 kg vs. 60.2 kg). The predicted intrinsic stiffness on the other hand, is affected by contributions from both laxities of passive tissues in trunk and active stiffness from background muscle activities, both of which have been demonstrated to be significantly smaller in females than males (Rozzi et al. 1999, Granata et al. 2002a, Granata et al. 2002b). A smaller intrinsic stiffness in females means a less stable trunk than an anthropometrically-matched male counterpart. This is consistent with findings of an earlier stability assessment experiment by Granata and Orishimo (2001) wherein it was shown that females demonstrated a significantly higher muscle co-activation to stabilize their trunk, particularly at higher task demands.

Both apparent mass and intrinsic stiffness increase with the level of muscle activity (Cholewicki et al. 2000, Gardner-Morse and Stokes 2001, Miller et al. 2010). As such, the reduction in apparent mass together with consistent levels of muscle activity found here indicate that exposure-induced decrements in intrinsic stiffness were due to alterations in passive mechanical trunk properties and not changes in background muscle activity (though activity of deeper trunk muscles was not monitored). The decrease in trunk apparent mass following exposure to trunk flexion could be related to changes in the dynamic response from wobbling of trunk soft tissues (Bazrgari et al. 2011). Such a response is in part affected by laxity of soft tissues, which increased following exposure to flexion in the present study. Increased soft tissue laxity results in a smaller inertial response (i.e., less mass is displaced) to a sudden perturbation. The predicted apparent mass in our model is a function of such inertial response, and which is the reason that apparent mass is much less than total trunk mass (i.e., ~ 50-60% of total body mass), and hence decreased following exposure to trunk flexion.

The observed effects of exposure to flexed postures on intrinsic trunk stiffness supports our hypotheses that both duration and external load increase the severity of changes, and that full recovery requires a duration longer than the initial disturbance time. While creep deformation values in the present study are comparable with earlier investigations, the overall 4-10% decrease in intrinsic stiffness may be an underestimation; measurements were performed in a neutral standing posture, which is associated with the least trunk stiffness (Parkinson et al.

2004, Shirazi-Adl 2006). In our earlier study (Hendershot et al. 2011), larger relative decreases (i.e., 10-20%) in intrinsic stiffness were found, in large part due to differences in experimental protocol (i.e., stress relaxation vs. creep deformation). Further, a new harness design was used here, which more tightly connected the thorax to the perturbing device and yielded higher estimates of intrinsic stiffness. Otherwise, absolute decreases in intrinsic stiffness in the present study (i.e., 682 (360) N/m) are comparable in magnitude to our earlier results (i.e., 936 (800) N/m).

Recovery of intrinsic stiffness varied directly but nonlinearly with exposure duration. Earlier studies have also reported a longer recovery period than the creep exposure time (McGill and Brown 1992, Rogers and Granata 2006, Shin and Mirka 2007), and further suggest a rapid but incomplete recovery of passive stiffness during the initial recovery period. Of note in our results is that trunk intrinsic stiffness continued to decrease during the first few minutes into the recovery period for cases with longer exposure duration. This is in contrast to patterns of intrinsic stiffness recovery following a stress-relaxation protocol, in which recovery was evident immediate after exposure and required a time comparable to exposure duration (Hendershot et al. 2011). It is unclear, though, what underlying mechanism is responsible for this difference in recovery from creep vs. load-relaxation exposures.

Reflexive trunk behavior

Reflexive trunk behaviors have been investigated using a variety of paradigms (sudden loading vs. unloading, or displacement control vs. force control perturbations), in different trunk postures (upright standing, flexed, supine), and in different loading directions (anteriorly vs. posteriorly). As such, there is a range in reported reflex behaviors (Cresswell et al. 1994, Wilder et al. 1996, Stokes et al. 2000, Granata et al. 2004, Rogers and Granata 2006, Moorhouse and Granata 2007, Sanchez-Zuriaga et al. 2010, Hendershot et al. 2011). Another important factor to consider when comparing the present with earlier results is that three of our reflexive measures (i.e., reflex gain and the timing and magnitude of maximum reflex force) were obtained from mechanical model-estimates of trunk reflexive force. Such measures thus represent a more global measure of trunk reflexive behavior, differing from EMG-driven estimates of trunk reflexive behaviors. Consistent with one earlier study (Granata et al. 2005), we found no effects of flexion exposure on EMG-driven muscle reflex delays. A more recent study (Sanchez-Zuriaga et al. 2010), though using a sudden-release paradigm in a flexed posture, reported a significant increase in such estimates of reflex delays after creep deformation. The minimum required strain in spinal ligaments to trigger reflex responses increases following ligamentous creep deformation (Le et al. 2009). Consistent with this, the timing of maximum reflex force, which was based on the overall trunk reflexive response, did increase following flexion here, suggesting that alterations in reflexive muscle behaviors might have occurred in muscle groups other than those monitored during the experiment. In the Le et al. (2009) study, however, spinal ligaments were stretched directly and reflex responses of the adjacent multifidus muscles were recorded, whereas here the whole trunk was flexed and a more global measure of trunk reflexive behavior was obtained.

Investigations using a feline model suggest a recovery pattern for reflexive muscle behavior following prolonged static and cyclic flexion-extension that includes an immediate and delayed hyper-excitability, with a period of decreased reflex response in between (Solomonow et al. 2003b). Our results confirmed an immediate hyper-excitability and subsequent decrease in the trunk reflexive behavior in humans. A delayed hyper-excitability period was not evident, though the recovery duration may have been insufficient. While an immediate reflexive hyper-excitability was observed, it did not appear to persist sufficiently to compensate for decreases in intrinsic trunk stiffness (i.e., during the early recovery period), hence supporting our third hypothesis (Fig 5). Following stress-relaxation exposures, this hyper-excitability continued even after complete recovery of trunk intrinsic stiffness (Hendershot et al. 2011). Since spinal ligament micro-damage has been suggested as a cause of disturbed neuromuscular behavior, the differences between our two sets of results may be attributed to differences in experimental protocols, in particular the different level of loadings that can affect the extent of such micro damage. Trunk flexion was achieved in our earlier work by raising the participant's legs while fixing the trunk upright. This resulted in a mean trunk passive resistance of 190 N, which dropped quickly to ~ 100 N due to stress relaxation, and which was substantially lower than the loading in the current work (i.e., constant trunk weight of ~ 400 N). As such, the disturbance to and recovery of reflexive behavior following prolonged trunk flexion appears to be highly dependent on the exposure conditions, in particular the history of trunk loading. In further support, Granata et al. (2005), using a similar experimental procedure, reported that muscle reflex gain increased following a single exposure to prolonged flexion, but that gain decreased after four separate exposures with rest between each (Rogers and Granata 2006).

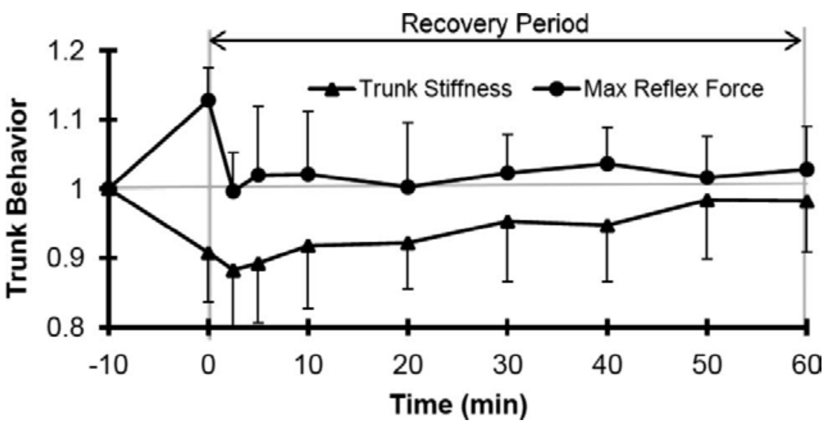

Figure 5: Disturbance and recovery of normalized trunk stiffness and maximum reflex force following exposure to 10 minutes of flexion. Error bars indicate standard deviations, and time = 0 indicates the end of the exposure period.

Muscle reflexes were significantly larger and faster in females than in males, likely due to higher levels of background muscle activity (Marras et al. 2002), and perhaps as compensation for lower contributions from intrinsic stiffness to the control of spinal stability. Females, though, demonstrated a faster recovery of reflex behavior post-exposure, to a level beyond initial values (Fig. 4). This pattern indicates a reduced control of spinal stability following prolonged flexion, and is consistent with the higher incidence rate of LBP in females (Pleis et al. 2009).

Implications of results

Occupations involving prolonged or frequent cycles of flexion are associated with higher risks of LBP, particularly when the trunk is flexed >60° for more than 5% of the working time (Hoogendoorn et al. 2000). Our findings suggest that this increased risk could be in part due to disturbances in trunk behaviors caused by flexion exposure. Spinal instability has been widely considered as an important cause of low back pain (Panjabi 2003). Stability of the spine is provided by force and stiffness contributions from the active trunk subsystem (i.e., voluntary and reflexive muscle responses), passive trunk subsystem (i.e., disc, ligaments, and passive muscle), and neuromuscular control subsystem (Panjabi 1992a). Passive trunk stiffness substantially decreased following flexion, and the recovery period exceeded that of the initial exposure. According to the model proposed by Panjabi (1992a), decreases in passive stiffness could be partially compensated by contributions from the active subsystem, through either increases in muscle activation / coactivation or increases in the muscle reflexive response (Moorhouse and Granata 2007, Brown and McGill 2008). However, such expected compensatory responses from the neuromuscular system were either absent (evidenced by constant background muscle activity immediately prior to perturbations) or of insufficient duration (evidenced by the rapid recovery of reflexive responses) to assure system stability. Such a situation can be expected to involve a higher risk of mechanical injury to the spine due to reduced stability. Since a rather long period is required for complete recovery of trunk behavior, which may not be practically feasible, interventions that change/reduce the exposure type (e.g., exposures that cause stress relaxation rather than creep deformation) or level (extent of flexion) may be more effective than, for example, changing work-rest cycles. Our findings also have potential implications in the design of exercise and rehabilitation programs, such as avoiding exposures that can adversely affect neuromuscular behavior (e.g., toe-touch stretching). Our participants, however, were university students and were exposed to only one bout of prolonged flexion. As such, future studies are needed to evaluate the effects of exposures among a broader population, longer-term exposures, and potential adaptive responses and behaviors.

Acknowledgments
This work was supported by an award (R01OH008504) from the Centers for Disease Control and Prevention (CDC). Its contents are solely the responsibility of the authors and do not necessarily represent the official views of the CDC. The authors thank Mr. Waldron and Mr. Vest for their assembly and maintenance of the testing apparatus. The authors dedicate this work to Dr. Kevin P. Granata whose original ideas formed the foundation for the current investigation.

References

Adams, M. A., and Dolan, P., 1996. Time-dependent changes in the lumbar spine's resistance to bending. *Clin Biomech*, 11 (4), 194-200.
Arjmand, N., Shirazi-Adl, A., and Bazrgari, B., 2006. Wrapping of trunk thoracic extensor muscles influences muscle forces and spinal loads in lifting tasks. *Clin Biomech*, 21 (7), 668-675.
Avela, J., Kyrolainen, H., and Komi, P. V., 1999. Altered reflex sensitivity after repeated and prolonged passive muscle stretching. *J Appl Physiol*, 86 (4), 1283-1291.
Baldwin, M. L., 2004. Reducing the costs of work-related musculoskeletal disorders: Targeting strategies to chronic disability cases. *J Electromyogr Kinesiol*, 14 (1), 33-41.
Bazrgari, B., Nussbaum, M. A., and Madigan, M. L., 2011 Estimation of trunk mechanical properties using system identification: Effects of experimental setup and modeling assumptions. *Comput Methods Biomech Biomed Engin*, (In press).
Bazrgari, B., Nussbaum, M. A., Madigan, M. L., and Shirazi-Adl, A., 2011. Soft tissue wobbling affects trunk dynamic response in sudden perturbations. *J Biomech*, 44 (3), 547-551.
Bazrgari, B., Shirazi-Adl, A., and Arjmand, N., 2007. Analysis of squat and stoop dynamic liftings: Muscle forces and internal spinal loads. *Eur Spine J*, 16 (5), 687-699.
BLS. 2009. Nonfatal occupational injuries and illnesses requiring days away from work: Bureau of Labor Statics.
Boschman, J. S., van der Molen, H. F., Sluiter, J. K., and Frings-Dresen, M. H., 2011. Occupational demands and health effects for bricklayers and construction supervisors: A systematic review. *Am J Ind Med*, 54 (1), 55-77.
Brown, S. H., and McGill, S. M., 2008. The intrinsic stiffness of the in vivo lumbar spine in response to quick releases: Implications for reflexive requirements. *J Electromyogr Kinesiol*.
Cholewicki, J., Simons, A. P. D., and Radebold, A., 2000. Effects of external trunk loads on lumbar spine stability. *J Biomech*, 33 (11), 1377-1385.

Cresswell, A. G., Oddsson, L., and Thorstensson, A., 1994. The influence of sudden perturbations on trunk muscle activity and intra-abdominal pressure while standing. *Exp Brain Res*, 98 (2), 336-341.
Dagenais, S., Caro, J., and Haldeman, S., 2008. A systematic review of low back pain cost of illness studies in the united states and internationally. *Spine J*, 8 (1), 8-20.
Dickey, J. P., McNorton, S., and Potvin, J. R., 2003. Repeated spinal flexion modulates the flexion-relaxation phenomenon. *Clin Biomech*, 18 (9), 783-789.
Fathallah, F. A., 2010. Musculoskeletal disorders in labor-intensive agriculture. *Appl Ergon*, 41 (6), 738-743.
Fathallah, F. A., Miller, B. J., and Miles, J. A., 2008. Low back disorders in agriculture and the role of stooped work: Scope, potential interventions, and research needs. *J Agric Saf Health*, 14 (2), 221-245.
Fowles, J. R., Sale, D. G., and MacDougall, J. D., 2000. Reduced strength after passive stretch of the human plantarflexors. *J Appl Physiol*, 89 (3), 1179-1188.
Gade, V. K., and Wilson, S. E., 2007. Position sense in the lumbar spine with torso flexion and loading. *J Appl Biomech*, 23 (2), 93-102.
Gallagher, S. 2008. Reducing low back pain and disability in mining: National Institute for Occupational Safety and Health (NIOSH).
Gardner-Morse, M. G., and Stokes, I. A., 2001. Trunk stiffness increases with steady-state effort. *J Biomech*, 34 (4), 457-463.
Granata, K. P., and Orishimo, K. F., 2001. Response of trunk muscle coactivation to changes in spinal stability. *J Biomech*, 34 (9), 1117-1123.
Granata, K. P., Padua, D. A., and Wilson, S. E., 2002a. Gender differences in active musculoskeletal stiffness. Part ii. Quantification of leg stiffness during functional hopping tasks. *J Electromyogr Kinesiol*, 12 (2), 127-135.
Granata, K. P., Rogers, E., and Moorhouse, K., 2005. Effects of static flexion-relaxation on paraspinal reflex behavior. *Clin Biomech*, 20 (1), 16-24.
Granata, K. P., Slota, G. P., and Bennett, B. C., 2004. Paraspinal muscle reflex dynamics. *J Biomech*, 37 (2), 241-247.
Granata, K. P., Wilson, S. E., and Padua, D. A., 2002b. Gender differences in active musculoskeletal stiffness. Part i. Quantification in controlled measurements of knee joint dynamics. *J Electromyogr Kinesiol*, 12 (2), 119-126.
Gupta, A., 2001. Analyses of myo-electrical silence of erectors spinae. *J Biomech*, 34 (4), 491-496.
Hendershot, B., Bazrgari, B., Muslim, K., Toosizadeh, N., Nussbaum, M. A., and Madigan, M. L., 2011. Disturbance and recovery of trunk stiffness and reflexive muscle responses following prolonged trunk flexion: Influences of flexion angle and duration. *Clin Biomech*, 26 (3), 250-256.
Hoogendoorn, W. E., Bongers, P. M., de Vet, H. C., Douwes, M., Koes, B. W., Miedema, M. C., et al., 2000. Flexion and rotation of the trunk and lifting at work are risk factors for low back pain: Results of a prospective cohort study. *Spine*, 25 (23), 3087-3092.
Jorgensen, M. J., Marras, W. S., Gupta, P., and Waters, T. R., 2003. Effect of torso flexion on the lumbar torso extensor muscle sagittal plane moment arms. *Spine J*, 3 (5), 363-369.
Katz, J. N., 2006. Lumbar disc disorders and low-back pain: Socioeconomic factors and consequences. *J Bone Joint Surg Am*, 88 Suppl 2, 21-24.
Kippers, V., and Parker, A. W., 1984. Posture related to myoelectric silence of erectores spinae during trunk flexion. *Spine*, 9 (7), 740-745.
Kuijer, P. P., Frings-Dresen, M. H., Gouttebarge, V., van Dieen, J. H., van der Beek, A. J., and Burdorf, A., 2011. Low back pain: We cannot afford ignoring work. *Spine J*, 11 (2), 164; author reply 165-166.

Le, B., Davidson, B., Solomonow, D., Zhou, B. H., Lu, Y., Patel, V., et al., 2009. Neuromuscular control of lumbar instability following static work of various loads. *Muscle Nerve*, 39 (1), 71-82.
Luo, X., Pietrobon, R., Sun, S. X., Liu, G. G., and Hey, L., 2004. Estimates and patterns of direct health care expenditures among individuals with back pain in the united states. *Spine*, 29 (1), 79-86.
Macintosh, J. E., Bogduk, N., and Pearcy, M. J., 1993. The effects of flexion on the geometry and actions of the lumbar erector spinae. *Spine*, 18 (7), 884-893.
Manchikanti, L., Singh, V., Datta, S., Cohen, S. P., and Hirsch, J. A., 2009. Comprehensive review of epidemiology, scope, and impact of spinal pain. *Pain Physician*, 12 (4), E35-70.
Marras, W. S., Davis, K. G., and Jorgensen, M., 2002. Spine loading as a function of gender. *Spine*, 27 (22), 2514-2520.
McGill, S. M., 2011. Letter to the editor regarding: "Causal assessment of occupational lifting and low back pain: Results of a systematic review" By wai et al. *Spine J*, 11 (4), 365.
McGill, S. M., and Brown, S., 1992. Creep response of the lumbar spine to prolonged full flexion. *Clin Biomech*, 7 (1), 43-46.
Miller, E., Bazrgari, B., Hendershot, B., Nussbaum, M. A., and Madigan, M. L. 2010. Dynamic response of the trunk to position perturbations – effects of gender, preload, and trunk angle. 34th annual meeting of the American Society of Biomechanics, Providence, Rhode Island, USA.
Moorhouse, K. M., and Granata, K. P., 2007. Role of reflex dynamics in spinal stability: Intrinsic muscle stiffness alone is insufficient for stability. *J Biomech*, 40 (5), 1058-1065.
Panjabi, M. M., 1992a. The stabilizing system of the spine. Part i. Function, dysfunction, adaptation, and enhancement. *J Spinal Disord*, 5 (4), 383-389; discussion 397.
Panjabi, M. M., 1992b. The stabilizing system of the spine. Part ii. Neutral zone and instability hypothesis. *J Spinal Disord*, 5 (4), 390-396; discussion 397.
Panjabi, M. M., 2003. Clinical spinal instability and low back pain. *J Electromyogr Kinesiol*, 13 (4), 371-379.
Parkinson, R. J., Beach, T. A., and Callaghan, J. P., 2004. The time-varying response of the in vivo lumbar spine to dynamic repetitive flexion. *Clin Biomech*, 19 (4), 330-336.
Pleis, J. R., Lucas, J. W., and Ward, B. W., 2009. Summary health statistics for u.S. Adults: National health interview survey, 2008. *Vital Health Stat 10*, 10 (242), 1-157.
Rogers, E. L., and Granata, K. P., 2006. Disturbed paraspinal reflex following prolonged flexion-relaxation and recovery. *Spine*, 31 (7), 839-845.
Rozzi, S. L., Lephart, S. M., Gear, W. S., and Fu, F. H., 1999. Knee joint laxity and neuromuscular characteristics of male and female soccer and basketball players. *Am J Sports Med*, 27 (3), 312-319.
Sanchez-Zuriaga, D., Adams, M. A., and Dolan, P., 2010. Is activation of the back muscles impaired by creep or muscle fatigue? *Spine*, 35 (5), 517-525.
Shin, G., and Mirka, G. A., 2007. An in vivo assessment of the low back response to prolonged flexion: Interplay between active and passive tissues. *Clin Biomech*, 22 (9), 965-971.
Shirazi-Adl, A., 2006. Analysis of large compression loads on lumbar spine in flexion and in torsion using a novel wrapping element. *J Biomech*, 39 (2), 267-275.
Solomonow, M., 2009. Ligaments: A source of musculoskeletal disorders. *J Bodyw Mov Ther*, 13 (2), 136-154.
Solomonow, M., Baratta, R. V., Banks, A., Freudenberger, C., and Zhou, B. H., 2003a. Flexion-relaxation response to static lumbar flexion in males and females. *Clin Biomech*, 18 (4), 273-279.
Solomonow, M., Baratta, R. V., Zhou, B. H., Burger, E., Zieske, A., and Gedalia, A., 2003b. Muscular dysfunction elicited by creep of lumbar viscoelastic tissue. *J Electromyogr Kinesiol*, 13 (4), 381-396.

Stokes, I. A., Gardner-Morse, M., Henry, S. M., and Badger, G. J., 2000. Decrease in trunk muscular response to perturbation with preactivation of lumbar spinal musculature. *Spine*, 25 (15), 1957-1964.
Twomey, L., and Taylor, J., 1982. Flexion creep deformation and hysteresis in the lumbar vertebral column. *Spine*, 7 (2), 116-122.
Wai, E. K., Roffey, D. M., Bishop, P., Kwon, B. K., and Dagenais, S., 2010. Causal assessment of occupational bending or twisting and low back pain: Results of a systematic review. *Spine J*, 10 (1), 76-88.
Weir, D. E., Tingley, J., and Elder, G. C., 2005. Acute passive stretching alters the mechanical properties of human plantar flexors and the optimal angle for maximal voluntary contraction. *Eur J Appl Physiol*, 93 (5-6), 614-623.
Wilder, D. G., Aleksiev, A. R., Magnusson, M. L., Pope, M. H., Spratt, K. F., and Goel, V. K., 1996. Muscular response to sudden load. A tool to evaluate fatigue and rehabilitation. *Spine*, 21 (22), 2628-2639.
Wilson, S. E., and Granata, K. P., 2003. Reposition sense of lumbar curvature with flexed and asymmetric lifting postures. *Spine*, 28 (5), 513-518.
Zhang, L., Huang, H., Sliwa, J., and Rymer, W., 1999. System identification of tendon reflex dynamics. *IEEE Transactions on Rehabilitation Engineering*, 7 (2), 193-203.

4. Aim 2: Influences of Flexion Angle and Duration on Load-Relaxation Effects on Neuromuscular Behaviors

Hendershot B, Bazrgari B, Muslim K, Toosizadeh N, Nussbaum MA, Madigan ML: [2011] Disturbance and recovery of trunk stiffness and reflexive muscle responses following prolonged trunk flexion: influences of flexion angle and duration. Clinical Biomechanics 26:250-256.

Abstract

Experimental studies suggest that flexed working postures reduce passive support of the spine, which could represent a significant risk factor for the development of occupational low back disorders. Neuromuscular compensations to reduced passive stiffness include increases in baseline activity or reflexive activation of trunk muscles. Yet, alterations and recovery of the synergy between active and passive tissues following prolonged flexion in humans are currently unknown. Twelve healthy participants were exposed to all combinations of two trunk flexion durations (2 and 16 min) and three flexion angles (33, 66, and 100% of individual flexion-relaxation angle). Load relaxation was recorded throughout exposures, whereas trunk stiffness and reflexive behaviors of the lumbar extensor muscles were investigated during dynamic responses to sudden perturbations. The magnitude of load relaxation increased with increasing flexion angle. Trunk stiffness decreased and reflex gains increased following flexion exposures; for both outcomes, acute changes were larger following exposure to increasing flexion angle. Reflex gains remained elevated one hour after exposure to maximum flexion. Exposure to prolonged trunk flexion changed trunk stiffness and reflex behavior in patterns consistent with epidemiological evidence linking such exposure with the risk of occupational low back disorders. Observed increases in reflex gains, at least among healthy individuals, may be a compensation for decreases in passive trunk stiffness following acute exposure to flexed postures. It remains to determine whether the neuromuscular system can similarly respond to accumulated disturbances in passive structures following exposure to repeated flexion tasks.

Introduction

Low back disorders (LBDs) remain the most common and debilitating occupational disorder (Andersson et al., 1996; Lawrence et al., 2006), accounting for ~40% of all reported occupational injuries (BLS, 2009). Epidemiological studies suggest that flexed working postures are a significant risk factor for LBDs (Kumar, 2001; Punnett and Wegman, 2004), and numerous experimental and modeling studies have been conducted to identify the underlying mechanism linking flexed working postures to the onset of LBDs. Recent reviews, however, indicate a lack of strong evidence supporting causality between manual material handling, bending/twisting and the development of low back pain, in large part due to study weaknesses (Roffey et al., 2010; Wai et al., 2010). In contrast, experimental results have demonstrated clear alterations in mechanical properties of the lower back following exposure to flexed postures. For example, trunk flexion range of motion has been reported to increase (2-6.5 degrees) following prolonged (10 - 60 minutes) flexion (McGill and Brown, 1992; Sanchez-Zuriaga et al., 2010; Shin and Mirka, 2007), indicating creep deformation of posterior trunk tissues. Similarly, a cadaveric study demonstrated a decrease in passive stiffness by nearly 42% following 5 minutes of sustained flexion (Adams and Dolan, 1996). Such viscoelastic behaviors in the trunk represent a reduction in passive support of the spine and would seem to require a compensatory adaptation of the active component of the neuromuscular system to maintain mechanical equilibrium and stability.

The neuromuscular system can compensate for reductions in passive stiffness with active stiffness. Active stiffness consists of both baseline muscle activation and reflexive activation of muscles, though the relative contribution of reflexive mechanisms to the control of spinal stability is controversial. While some investigators suggest that the spinal column may be sufficiently stabilized against sudden unexpected loads by baseline activation only (e.g., Stokes et al., 2000), others argue that reflexive muscle activation is also required to adequately control spinal stability and prevent harmful spinal displacements (Brown and McGill, 2008; Moorhouse and Granata, 2007). Alterations in baseline trunk muscle activities following exposure to flexed posture has been confirmed by a delayed occurrence of the flexion-relaxation phenomenon (Olson et al., 2004; Shin and Mirka, 2007; Solomonow et al., 2003a), indicating that muscles remain active over a larger range of trunk flexion to compensate for reduced passive stiffness. Reflexive activation of trunk muscles is also affected by prolonged flexed postures (Granata et al., 2005b; Rogers and Granata, 2006; Sanchez-Zuriaga et al., 2010; Solomonow, 2004). Passive stretch of skeletal muscles during prolonged flexion can reduce muscle spindle excitability thereby inhibiting reflexive muscle response (Avela et al., 1999; Rosenbaum and Hennig, 1995). Creep deformation and load relaxation of spinal ligaments have been shown to be associated with an immediate decrease in reflex behavior (Solomonow et al., 2000; Solomonow et al., 1999), while others have reported immediate or delayed increases in reflexive muscle responses following passive stretching (Granata et al., 2005b; Solomonow et al., 2003b). Though it appears that alterations in muscle activity following prolonged flexed posture is a compensatory response to reduced spinal stiffness, it is currently unclear whether changes in reflexive behavior is similarly a compensatory response, or perhaps another impairment to the control of spinal stability.

Recent evidence suggests that disturbances to the neuromuscular control of spinal stability may persist for extended periods after exposure to flexed postures (Rogers and Granata, 2006; Solomonow et al., 2003b). This may be due, in part, to the fact that recovery of passive stiffness following prolonged flexion requires more time than the initial exposure due to creep deformation or load relaxation (Gedalia et al., 1999; McGill and Brown, 1992; Shin and Mirka, 2007; Solomonow et al., 2003b). Moreover, repetitive prolonged flexion with insufficient recovery time causes a significant accumulative disturbance (Solomonow et al., 2003b). Although both a slow recovery of neuromuscular behavior and a significant accumulation of disturbance have been shown in animal models, it remains to be investigated in humans. We have developed a new method to quantify the active and passive components of the neuromuscular system that contribute to spinal stability. The goal of the present work was to use this method to investigate the effects of prolonged trunk flexion on these active and passive components. We hypothesized that: (1) the severity of alterations in active and passive components increases with flexion duration and angle, and (2) recovery is influenced by the initial severity of alterations in system behavior.

Methods

Participants

Twelve healthy young adults with no self-reported history of low-back pain participated, after completing informed consent procedures approved by the Virginia Tech Institutional Review Board. Participants included six males with mean (SD) age, stature, and body mass of 23 (4) yr, 180.3 (6.8) cm, and 75.3 (10.8) kg, respectively; corresponding values for the six females were 22 (3) yr, 166.1 (7.9) cm, and 60.1 (5.5) kg. A relatively young set of participants was included to avoid potential influences related to age. All participants reported being free of current or recent injuries, illnesses, musculoskeletal disorders, and other health-related aspects that might have influenced the results.

Experimental Design and Procedures

A repeated measures design was used, in which several measures of the trunk neuromuscular system were obtained prior to, during, and following exposures to prolonged trunk flexion. There were six different exposure conditions, which differed in duration (two levels) and the extent of flexion (three levels), and with the latter specified relative to each participant's flexion-relaxation (FR) angle. Each exposure condition was tested at a similar time on separate days with a minimum of 72 hours between consecutive tests. To reduce the potential for order-related confounding effects, the order of conditions was specified using balanced Latin Squares (i.e., two 6x6 squares, one for each gender group).

At the beginning of each experimental session, three trials were performed to record the lumbar angle at which FR occurred. Each trial involved relatively slow movement from a standing upright posture to full trunk flexion (~5 sec) and a return to a standing upright posture (~5 sec). Lumbar flexion angle was estimated using two 6 DOF inertial motion units (IMUs: Xsens Technologies XM-B-XB3, Enschede, Netherlands) placed over the T10 vertebral process and the superior aspect of the sacrum (S1). Electromyographic (EMG) activity of the erector spinae muscles was recorded bilaterally using bipolar Ag/AgCl surface electrodes placed over the muscle belly at the L3 level (Larivière et al., 2009). Prior to applying electrodes, the skin was prepared using abrasion and cleaned with alcohol, and inter-electrode impedance was maintained below 10KW. Raw EMG signals were preamplified (x100) near the collection site, bandpass filtered (10-500Hz), amplified and converted to RMS in hardware (Measurement Systems Inc., Ann Arbor, MI, USA). Kinematic data were sampled at 100 Hz and EMG at 1000 Hz. FR angle was determined from data obtained during trunk flexion, as the lumbar angle when myoelectric silence occurred in the averaged L3 erector spinae (Solomonow et al., 2003a).

After determining FR angle, participants subsequently stood in a rigid metal frame and adjustable straps were used to restrain the pelvis and lower limbs. While in the frame, participants wore a shoulder harness that was connected by a rigid rod at the T8 level to a servomotor (Kollmorgen AKM53K, Radford, VA, USA) attached to the frame (Figure 1). The frame had an adjustable foot support that was set such that the pelvis and lower extremities could be rotated about a transverse axis at the approximate sagittal-plane location of the L5/S1 joint (Figure 1). Rotating the pelvis and lower extremities upward (instead of the trunk downward) to control trunk flexion angle avoided confounding influences of changes in muscle recruitment in response to gravity requirements or muscle fatigue.

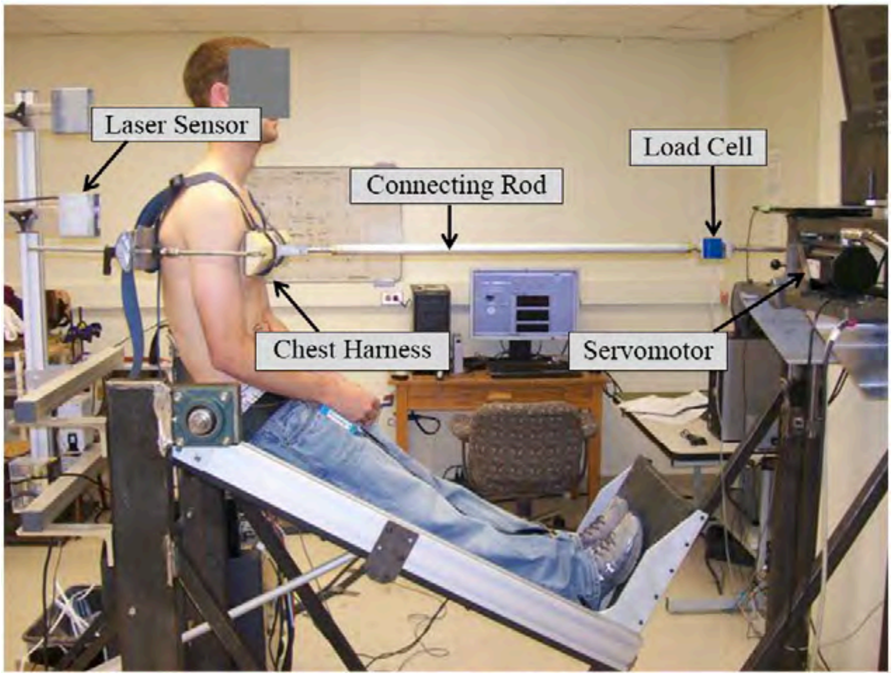

Figure 1: Experimental set up, demonstrating a participant in a flexed posture (100% of FR angle).

During each session, participants were exposed to one of the six combinations of two trunk flexion durations (2 and 16 min) and three flexion angles (33, 66, and 100% of individual FR angle). These combinations of duration and angle were intended to represent a range of potential occupational exposures. Across participants, mean FR angle was 65.2 (7.6) ˚, and the mean flexion angles used for testing were 21.5 (2.5), 43.7 (5.1), and 65.2 (7.6) ˚. Rotating the lower extremities to achieve a specific flexion angle induces stretch in the passive tissues of the lower back, causing an extension moment on the spine and a resulting tensile reaction force in the connecting rod. These reaction forces were sampled at 1000 Hz throughout the exposures, using a load cell (Interface SM2000, Scottsdale, AZ, USA) in series with the connecting rod (Figure 1). Mean tensile forces at the beginning of each flexion exposure were 32.2 (14.9), 54.6 (28.3), and 190.5 (84.8) N for the 33, 66, and 100% exposures, respectively. Changes in these forces represent the load relaxation behavior of the posterior trunk tissues.

Sudden Perturbations

Pre- and post-exposure measures of trunk behavior were obtained using a sudden-perturbation paradigm as described below. These measures were collected while participants were in an upright posture (i.e., trunk flexion angle = 0˚). The typical delay between the end of exposure and the first post-exposure measurement was ~30 sec. Participants then maintained the upright posture while strapped to the frame for 60 min to assess post-exposure recovery. Specifically, recovery measures were obtained 2.5, 5, and 10 min after exposure, and every 10 min thereafter until 60 min post-exposure. The spacing of these post-exposure measurements was designed to capture the exponential behavior of the recovery process, and the 10 min intervals toward the end were based on a previous study (Gedalia et al., 1999).

A sudden-perturbation paradigm was used to quantify the behavior of active and passive neuromuscular components as in earlier studies (e.g., Brown and McGill, 2009; Hodges et al., 2009; Moorhouse and Granata, 2007). A sequence of 12 anterior-posterior position perturbations (± 5 mm), generated by the servomotor rotations and transferred to the trunk via the rod-harness assembly over a ~ 30 s duration. Pseudorandom delays between each perturbation were used to prevent anticipation of perturbation timing by the participants and hence reduce potential confounding from variations in anticipatory muscle activation. Rapid perturbations were used to allow separation of baseline activation responses and reflexive responses, as described below (Figure 2). Each perturbation was completed within 40 ms, which is less than typical erector spinae reflex delays (Granata et al., 2004; Hwang et al., 2008).

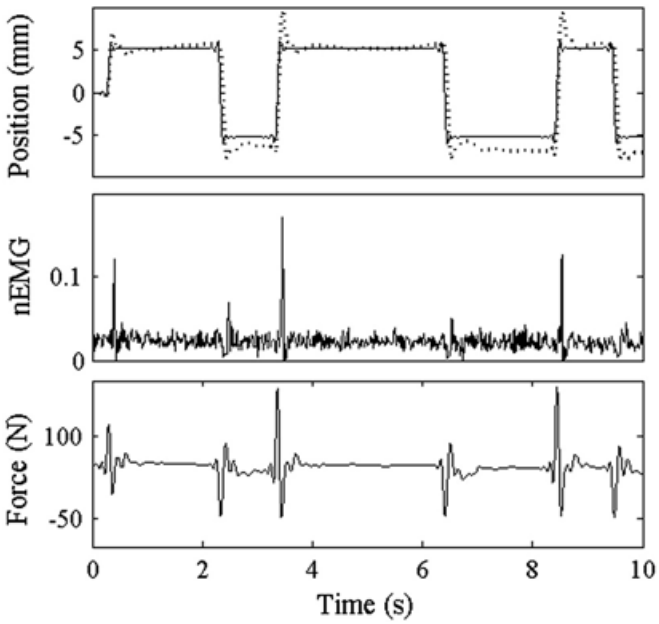

Figure 2: Representative pre-exposure data obtained during several anterior-posterior perturbations (a 10-sec subset is illustrated). (Top) Pseudorandom sequence of trunk positions as indicated by the servomotor encoder (solid line) and measured displacement of T10 from the laser sensor (dotted line). Positive values indicate anterior perturbations. (Middle) Mean of bilateral normalized EMG (nEMG) of the L3 erector spinae, showing reflex spikes following anterior perturbations. (Bottom) Driving force measured from the in-line load cell (positive values indicate tension in the rigid rod connecting the motor to the participant).

During perturbations, trunk motion was measured using both a high-accuracy encoder (resolution ± 60 arcsec) attached to the servomotor shaft, and a high-accuracy (± 0.5 μm, response time = 0.2 msec) CCD laser displacement sensor (Keyence LK-G 150, Osaka, Japan) attached to the frame (Figure 1). The latter was targeted at the midline of the dorsal aspect of the trunk, just above the harness. Driving forces during perturbations were measured using the in-line load cell. Load cell, motor encoder, and laser sensor data were sampled at 1000 Hz, then low-pass filtered using a 7th-order, bidirectional, Butterworth filter with a 10 Hz cutoff frequency (Bazrgari et al., 2010b). Muscle activity (EMG) was measured and processed as described earlier, with additional electrodes placed bilaterally over the L1 erector spinae (Larivière et al., 2009) and the rectus abdominus and external oblique (Granata et al., 2005a).

For ~3 sec prior to and during the perturbation sequences, participants maintained a constant baseline extensor effort. From several maximum voluntary isometric trunk extensions, conducted in each experimental session following trials to determine FR angle, peak extensor RMS EMG activity was obtained. The target baseline effort was set to 10% of the maximum voluntary activation level in the bilateral L3 erector spinae, and during perturbations real-time visual feedback of the target was provided. Preliminary work indicated that using EMG for visual feedback (rather than force) provided more consistency in preload efforts. Further, control of preload using extensor EMG was expected to minimize potential influences of alterations in muscle recruitment (e.g. changes in co-contraction). Specifically, since both the extensor activity and net joint moment were consistent, it was presumed that only minimal variability existed in other agonists or antagonists (the latter of particular concern, due to effects on effective joint stiffness). Mean (SD) baseline preload efforts were 69 (14) N for males and 64 (10) N for females.

Trunk measures

Initially, the latency of reflexive muscle response (t_d) following each perturbation was determined as the time delay between displacement onset and the onset of erector spinae muscle reflex response, the latter identified as peaks exceeding two standard deviations above mean activity prior to the perturbations (Granata et al., 2004; Zhang et al., 1999). Trunk measures were then investigated by quantifying the trunk dynamic response to the applied perturbations in two separate time windows. Trunk dynamic properties within the first time window have been referred as "intrinsic" properties in earlier investigations (Brown and McGill, 2009; Moorhouse and Granata, 2007), influenced by passive stiffness and baseline muscle activity (i.e., the 10% MVC preload). This window began from the onset of an anterior perturbation and ended at the reflex onset of the erector spinae musculature. The second time window had components of both intrinsic properties and reflexive neuromuscular response, and had a length of ~150 ms starting from reflex onset.

Intrinsic properties were identified by relating measured trunk kinematics (i.e., horizontal movement at the T8 level obtained from the laser sensor and motor encoder) to trunk kinetics (i.e., reaction force obtained from the load cell) during the first time window and by modeling the trunk as a single degree-of-freedom mass-spring-damper system with three parameters: effective mass (m), damping (c), and stiffness (k). An extra mass-spring-damper element was included in the model to account for confounding from dynamic properties of connecting elements, specifically the connecting rod, harness, and soft tissues such as skin and padding at the trunk-harness interface (Bazrgari et al., 2010a, 2010b). Model parameters were obtained by minimizing the error between measured and model-predicted driving forces, using the following system of equations:

$$F(u,t) = \begin{Bmatrix} F_1 \\ F_2 \end{Bmatrix} = \begin{bmatrix} 1.5 & 0 \\ 0 & m_2 \end{bmatrix} \begin{Bmatrix} \ddot{u}_1 \\ \ddot{u}_2 \end{Bmatrix} + \begin{bmatrix} c_1 & -c_1 \\ -c_1 & c_1 + c_2 \end{bmatrix} \begin{Bmatrix} \dot{u}_1 \\ \dot{u}_2 \end{Bmatrix} + \begin{bmatrix} k_1 & -k_1 \\ -k_1 & k_1 + k_2 \end{bmatrix} \begin{Bmatrix} u_1 \\ u_2 \end{Bmatrix}$$

where subscripts *1* and *2* refer to the connecting elements and the trunk, respectively, F is an external force applied to a degree of freedom, and m, c, and k respectively denote mass, damping, and stiffness. The constant *1.5* represents the mass (kg) of the connecting elements and u (u_1 from motor encoder and u_2 from laser sensor) and its derivatives denote the displacement, velocity, and acceleration of the system's degrees of freedom. For this analysis, trunk damping (c_2) was forced to zero (cf., Gardner-Morse and Stokes, 2001) so that any alterations in trunk behavior could be represented by changes in trunk stiffness (k_2); earlier work also suggested that trunk damping may be negligible (Cholewicki et al., 2000). Using this approach, trunk stiffness (k_2) includes the resistance from both passive tissue and baseline muscle activation. Model parameters were estimated using a least-squares curve fit in MATLAB™ (MathWorks, Natick, MA, USA). Of the 12 anteriorly-directed perturbations that occurred during each testing sequence, the perturbation yielding the highest bivariate coefficient of correlation between measured and model-predicted driving forces was used for subsequent analyses. Correlation values for these ranged from 0.958 - 0.994 and were consistent across all six exposure conditions.

Trunk reflexive behaviors were characterized by reflex gain (G_R). Reflex gains were first estimated by subtracting the estimated intrinsic force contribution from the total measured trunk response (reaction force from the load cell) during the second time window (i.e., after the reflex delay). This difference in force was then correlated to time-shifted (equal to predicted reflex delay) trunk velocity, so that both were aligned in time (Moorhouse and Granata, 2007). Therefore, reflex gains here are a measure of the magnitude of the reflex response with respect to trunk velocity. Larger reflex gains indicate a greater reflex response.

Analyses

Load relaxation in passive trunk tissues due to flexion exposures was characterized as the difference in force measured by the load cell from the beginning to end of each exposure period. Such differences were also determined for the total RMS EMG activity (summed across all muscles) during the 3-second baseline extensor effort prior to each perturbation sequence. Changes in total RMS EMG activity were used as a surrogate for any alterations in agonist and antagonist muscle activity (McCook et al., 2009); specifically, a post-exposure increase in total muscle activity was assumed to indicate increases in co-contraction and spinal load. Pre-exposure differences in trunk stiffness, reflex delay, and reflex gain between genders were evaluated using unpaired *t*-tests. Post-exposure measures of trunk stiffness (k_2) and reflex gains (G_R) were normalized to pre-exposure values [(post-pre)/pre]. Acute effects of flexion angle, duration, and gender were assessed using mixed-factor, repeated measures analyses of variance (ANOVA), and a repeated measures MANOVA was used to assess the effects of these same factors over the recovery period. No significant deviations from parametric model assumptions were evident. Where relevant, post-hoc pairwise comparisons were performed using Tukey's HSD. Data from one trial of one participant (male, 16 min. of exposure to 33% flexion angle) were excluded from these analyses due to measurement errors. All analyses were done using JMP™ (Version 8, SAS Institute Inc., Cary, NC), and statistical significance was concluded when $P<0.05$. Summary values are reported as means (SD).

Results

Pre-exposure

Pre-exposure trunk stiffness (k_2) was higher ($t_{(70)} = 3.52$; $P=0.0008$) among males than females, at 4912 (1632) and 3540 (1677) N/m, respectively. Muscle reflex delays (t_d) were shorter ($t_{(70)} = -2.73$; $P=0.008$) among males [62.0 (3.2) ms] than females [64.6 (5.8) ms]. Pre-exposure reflex gains (G_R) did not differ ($t_{(70)} = -0.73$; $P=0.47$) between males [929 (245) Ns/m] and females [972 (260) Ns/m].

Post-exposure

Load relaxation increased with increasing flexion angle ($F_{(2,41)} = 16.62$; $P<0.0001$), tended to increase with exposure duration ($F_{(1,41)} = 3.08$; $P=0.086$), but did not differ between genders ($F_{(1,9)} = 0.14$; $P=0.71$; Figure 3).

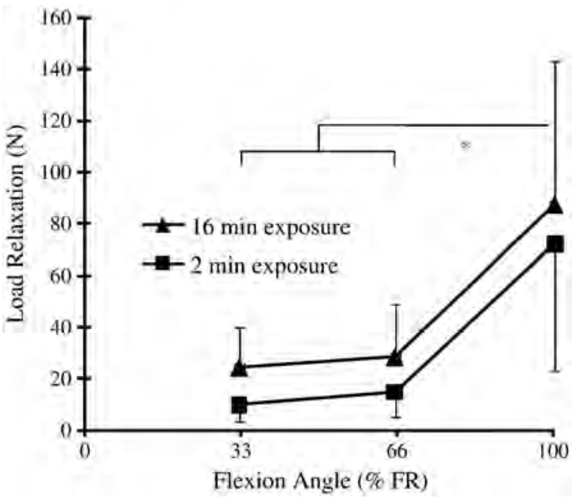

Figure 3: Effects of flexion angle and duration on trunk load relaxation. Error bars indicate standard deviations, and results from post-hoc pairwise comparisons are indicated by brackets (* = significant difference between flexion angles).

Across all exposure conditions, there was a significant ($t_{(70)}$ = -3.80; P=0.0003) decrease in trunk stiffness of 372 (824) N/m. This decrease was influenced by flexion angle ($F_{(2,44)}$ = 11.28; P=0.0001), with larger decreases following exposure to increased angles (Figure 4). Specifically, trunk stiffness decreased by 1.6 (11.5), 7.8 (15.8), and 24.5 (12.3) % following exposure to the 33, 66, and 100% flexion angles, respectively. Decreases in trunk stiffness were not different between the two exposure durations ($F_{(1,44)}$ = 0.47; P=0.50), and females had a 6.8 % larger decrease in trunk stiffness than males ($F_{(1,10)}$ = 5.59; P=0.04). Reflex delays were unaffected by trunk flexion angle ($F_{(2,44)}$= 0.01; P=0.99) or duration ($F_{(1,44)}$ = 0.64; P=0.43), with respective pre- and post-exposure values of 63.3 (4.9) and 63.4 (4.3) ms. Reflex gains significantly increased by 76.9 (133.2) Ns/m across all exposure conditions ($t_{(70)}$ = 4.87; P<0.0001). Similar to trunk stiffness, this effect differed between flexion angles ($F_{(2,44)}$ = 16.89; P<0.0001; Figure 4), but was not affected by exposure duration ($F_{(1,44)}$ = 0.95; P=0.34) or gender ($F_{(1,10)}$ = 1.02; P=0.34). Reflex gains increased by 1.4 (8.5), 9.4 (12.2), and 20.9 (12.7) % after exposure to 33, 66, and 100% flexion angles, respectively. Baseline trunk muscle activity (total RMS across all muscles) was not affected by flexion angle ($F_{(2,49)}$ = 1.07; P=0.35), duration ($F_{(1,49)}$ = 0.54; P=0.47), or gender ($F_{(1,10)}$ = 3.58; P=0.09), with respective pre- and post-exposure values of 0.44 (0.096) and 0.45 (0.095) V.

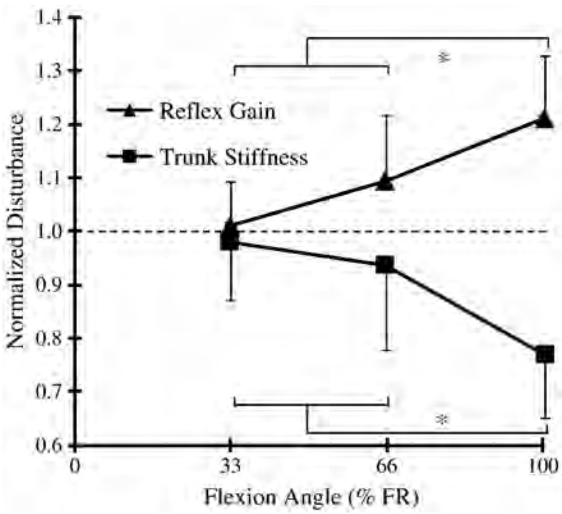

Figure 4: Effects of flexion angle on normalized changes in trunk stiffness (k_2) and reflex gain (G_R). Error bars indicate standard deviations, and results from post-hoc pairwise comparisons are indicated by brackets (* = significant difference between flexion angles).

Recovery

Consistent trends were evident in the patterns of recovery of trunk stiffness and reflex gain following exposure to maximum flexion (100% FR) after both 2 and 16 minutes of exposure (Figure 5), and MANOVA indicated no differences with respect to exposure duration ($F_{(1,18)}$ = 0.004; P=0.79). Recovery patterns were inconsistent following exposure to the smaller flexion angles, and hence no statistical analyses were undertaken. Following exposure to 100% FR, there was a gender effect on recovery of trunk stiffness ($F_{(1,20)}$ = 0.27; P=0.03). As previously noted, females had a 6.8 % larger decrease in stiffness following flexion exposures. Qualitatively, females recovered at a faster rate, reaching pre-exposure levels at roughly the same time as males. There was no gender effect on reflex gain ($F_{(1,18)}$ = 0.002; P=0.87).

Reflex delays remained constant throughout the recovery period and did not differ between exposure durations ($F_{(1,21)}$ = 0.008; P=0.68).

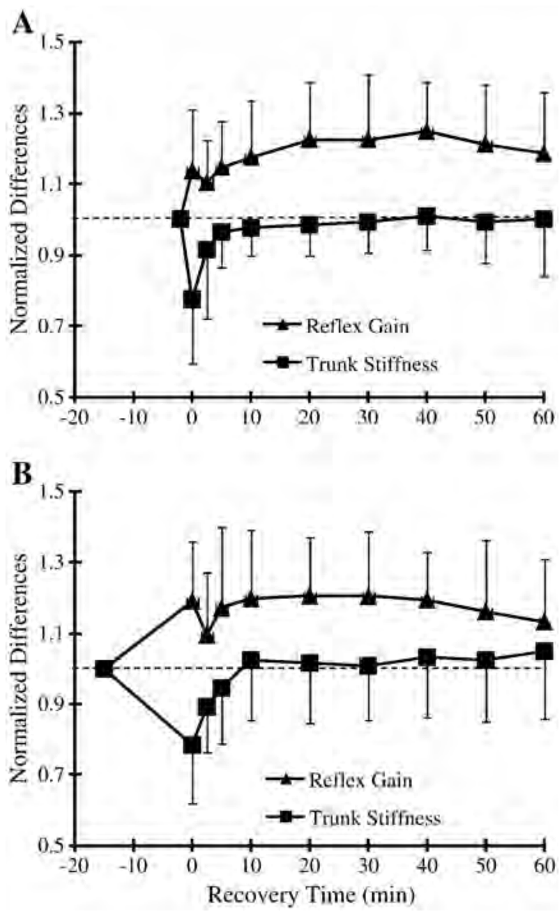

Figure 5: Recovery of trunk stiffness (k_2) and reflex gain (G_R) following exposure to maximum flexion for 2 (A) and 16 (B) minutes. Post-exposure values are normalized to those obtained pre-exposure. Error bars indicate standard deviations and time = 0 indicates the end of the exposure period.

Discussion

Passive trunk stiffness, as measured by changes in load relaxation force, decreased by 38.8 (10.4) % across all six exposure conditions and was comparable to a 42% reduction reported earlier following 5 minutes of sustained flexion (Adams and Dolan, 1996). Exposure-induced decreases in passive stiffness (i.e., load relaxation) occurred in an exponential manner, with most (90%) of the decrease occurring in the first two minutes of flexion exposure (Toosizadeh et al., 2010). This relatively rapid response accounts for the general similarity in measured responses between the two flexion durations.

Trunk stiffness measured during the current sudden perturbations represents the overall contribution of passive trunk tissues and baseline muscle activation. Trunk stiffness (k_2) decreased more following exposure to larger trunk flexion angles, yet no differences were found in baseline activation levels. Hence, the observed decrease in trunk stiffness is likely a result of decreases in passive stiffness. Similar decreases in passive stiffness have been inferred in earlier studies based on an increase in flexion range of motion and a delayed onset of myoelectric silence (McGill and Brown, 1992; Rogers and Granata, 2006; Shin and Mirka, 2007). Such a reduction in trunk stiffness represents a disturbance to mechanical equilibrium and overall trunk stability, and may impose a higher injury risk (Hoogendoorn et al., 2000).

Decreases in trunk stiffness were on the order of 10-20% across all experimental conditions, and may be an underestimation since all measurements were made in the upright posture (i.e., flexion angle = 0°). Contributions from passive stiffness are considerably lower near the neutral posture, increasing nonlinearly toward the end range of motion in both flexion and extension (Brown and McGill, 2008). Therefore, we expect that larger decreases in trunk stiffness would have been found had all measurements been made in a flexed posture (i.e., closer to end range of motion). This was not done, however, to avoid potential confounding from the cyclic flexion motions that would be required to measure and return to upright standing throughout the experiment.

While current literature relating to spinal stability places a large emphasis on trunk stiffness as a major feedback control mechanism, trunk damping has recently been suggested as a means for better understanding spinal stability and neuromuscular control (Reeves and Cholewicki, 2010). Initial analyses revealed that our model predicted the dynamic response of the trunk to be dominated by damping. To be consistent with existing literature and facilitate comparisons, we forced trunk damping (c_2) to zero. This did not affect the overall results, in terms of the effects of trunk flexion angle and duration, but simply transferred the dominating response from damping to stiffness. Further, despite forcing damping to zero, estimated effective trunk mass (m_2) was highly correlated ($r^2 = 0.9$) with body mass. It is important to note that the mean quality of model predictions across all subjects and trials was 98.2 % and 98.6 % for the constrained (i.e., damping = 0) and non-constrained (i.e., non-zero damping) modeling approaches, respectively. Accordingly, both modeling approaches were able to sufficiently represent the passive viscoelastic resistance of the trunk. However, some caution is warranted when interpreting the relative contribution of elastic and viscous components here and in earlier work, since the models used represent potential oversimplifications of the spine and the predicted properties may thus not be the best physical representation of the system.

Exposure to increasing flexion angles led to larger increases in reflex gain. This suggests that alterations to the reflexive response are compensatory for increasing reductions in passive stiffness (Figure 4). Earlier investigations using a feline model (Solomonow, 2003b) and humans (Granata et al., 2005b) have reported similar compensatory reflex responses. The neuromuscular system can compensate for flexion-induced decreases in passive stiffness by increasing reflexive responses and/or baseline muscle activation. The former represents the ability of the trunk neuromuscular system to respond quickly to postural displacements through sensory stretch-receptor reflex loops, which has been suggested to provide ~ 40% of total effective trunk stiffness (Moorhouse and Granata, 2007). The latter, and perhaps more metabolically unfavorable, is to increase overall muscle activation/coactivation, and evidence suggests that increasing muscle activity substantially increases trunk stiffness (Gardner-Morse and Stokes, 2001; Moorhouse and Granata, 2007). Our results suggest that increased reflex gains following exposure to prolonged trunk flexion act as a metabolically-efficient compensation for decreases in trunk stiffness, and perhaps also serve to protect stretched ligaments from further micro-damage until recovery can occur (Solomonow, 2004).

Understanding the recovery from such alterations in passive and reflexive trunk neuromuscular control has potential occupational implications, such as in designing work-rest cycles or job rotation schedules. In contrast to earlier studies that show recovery of passive stiffness takes longer than the initial exposure duration (McGill and Brown, 1992; Shin and Mirka, 2007), the current results suggest that recovery requires roughly the same duration as the exposure (Figure 5). This may be explained by differences in methodology, however, in that others have measured recovery near the end range of motion while we obtained all measurements in an upright posture. At the same time, reflex gains remained elevated for a longer period, perhaps to ensure full recovery of passive stiffness. Specifically, reflex gains remained elevated one hour after exposures to maximum flexion though returning toward initial values (Figure 5). It is unclear how much time would be required for full recovery or whether such prolonged increases in reflex gains are detrimental to the spine/trunk.

The experimental protocol was designed to separate the effects of flexion angle from gravity-imposed trunk moment and the resultant muscle activity. This was achieved by raising the legs, rather than flexing the trunk, and as a result involved stress relaxation of passive tissues in the spine. In contrast, typical occupational tasks involve trunk flexion, wherein the bending moment is maintained and the passive tissues undergo creep deformation. Despite this limitation, our results are considered useful toward understanding potential mechanisms involved in some cases of occupational low back disorders, and future studies are planned to assess the influence of creep deformation of the human trunk (e.g., resulting from static and intermittent load handling).

In summary, the current findings begin to identify how the neuromuscular system adapts to viscoelastic changes in the trunk following prolonged flexion in healthy individuals. Some evidence of dose-response relationships was apparent here, between the angle and duration of trunk flexion and resulting alterations in neuromuscular behaviors. These are qualitatively consistent with the epidemiological results of Hoogendoorn et al. (2000), and suggest potential pathophysiological mechanisms and future strategies to control exposures. Regarding such mechanisms, earlier studies indicate that patients with low-back pain exhibit higher muscle activities prior to sudden perturbations (Stokes et al., 2006) and higher levels of co-activation (Radebold et al., 2000) when compared to healthy controls, while delayed reflexive responses may be a risk factor for the development of low-back pain or injury (Cholewicki et al., 2005). This evidence, along with the current results, suggest that a lack of efficient reflexive control may require the neuromuscular system to make a metabolically and mechanically unfavorable decision to provide a constant increase in muscles activities to assure spinal stability. Such suboptimal performance of the neuromuscular system in healthy individuals has been suggested as a potential cause of LBDs (Cholewicki et al., 2005). Of note, reflex gains increased following prolonged trunk flexion, perhaps to compensate for decreases in trunk stiffness, and remained elevated for some time after exposure, perhaps to ensure full recovery of passive stiffness. Alterations in and recovery of the synergy between active and passive trunk behavior following flexed postures requires further study, particularly in older individuals and those with low-back pain. It also remains to determine if the neuromuscular system can efficiently respond to accumulated disturbances to passive structures resulting from repetitive trunk flexion-extension, such as is commonly encountered during occupational manual material handling tasks.

Acknowledgments
This work was supported by Cooperative Agreement Number R01OH008504 from the Centers for Disease Control and Prevention (CDC). Its contents are solely the responsibility of the authors and do not necessarily represent the official views of CDC. The authors thank Mr. Waldron and Mr. Vest for their assembly and maintenance of the testing apparatus, and Mr. Seung-Yeun Han for his assistance with data collection. The authors dedicate this work to Dr. Kevin P. Granata whose original ideas formed the foundation for the current investigation.

References
Adams, M. A., and Dolan, P., 1996. Time-dependent changes in the lumbar spine's resistance to bending. Clin. Biomech. 11, 194-200.

Andersson, E. A., Oddsson, L. I. E., Grundström, H., Nilsson, J., and Thorstensson, A., 1996. Emg activities of the quadratus lumborum and erector spinae muscles during flexion-relaxation and other motor tasks. Clin. Biomech. 11, 392-400.
Avela, J., Kyrolainen, H., and Komi, P. V., 1999. Altered reflex sensitivity after repeated and prolonged passive muscle stretching. J. Appl. Physiol. 86, 1283-1291.
Bazrgari, B., Nussbaum, M. A., and Madigan, M. L., 2010a. Effects of experimental setup and modeling assumptions on predicted trunk properties using a system identification method. 34th annual meeting of the American Society of Biomechanics, Providence, Rhode Island, USA. (http://www.asbweb.org/conferences/2010/abstracts/44.pdf)
Bazrgari, B., Nussbaum, M. A., and Madigan, M. L., 2010b. Estimation of trunk mechanical properties using system identification: Effects of experimental setup and modeling assumptions. Submitted to the J. Biomech. Eng.
BLS, 2009. Nonfatal occupational injuries and illnesses requiring days away from work (Report No. USDL-09-1454). U. S. Department of Labor, Washington, D.C.
Brown, S. H. M., and McGill, S. M., 2008. How the inherent stiffness of the in vivo human trunk varies with changing magnitudes of muscular activation. Clin. Biomech. 23, 15-22.
Brown, S. H. M., and McGill, S. M., 2009. The intrinsic stiffness of the in vivo lumbar spine in response to quick releases: Implications for reflexive requirements. J. Electromyogr. Kinesiol. 19, 727-736.
Cholewicki, J., Silfies, S. P., Shah, R. A., Greene, H. S., Reeves, N. P., Alvi, K., et al., 2005. Delayed trunk muscle reflex responses increase the risk of low back injuries. Spine 30, 2614-2620.
Cholewicki, J., Simons, A. P. D., and Radebold, A., 2000. Effects of external trunk loads on lumbar spine stability. J. Biomech. 33, 1377-1385.
Gardner-Morse, M. G., and Stokes, I. A. F., 2001. Trunk stiffness increases with steady-state effort. J. Biomech. 34, 457-463.
Gedalia, U., Solomonow, M., Zhou, B.-H. E. E., Baratta, R. V., Lu, Y., and Harris, M., 1999. Biomechanics of increased exposure to lumbar injury caused by cyclic loading: Part 2. Recovery of reflexive muscular stability with rest. Spine 24, 2461-2467.
Granata, K. P., Lee, P. E., and Franklin, T. C., 2005a. Co-contraction recruitment and spinal load during isometric trunk flexion and extension. Clin. Biomech. 20, 1029-1037.
Granata, K. P., Rogers, E., and Moorhouse, K., 2005b. Effects of static flexion-relaxation on paraspinal reflex behavior. Clin. Biomech. 20, 16-24.
Granata, K. P., Slota, G. P., and Bennett, B. C., 2004. Paraspinal muscle reflex dynamics. J. Biomech. 37, 241-247.
Hodges, P., van den Hoorn, W., Dawson, A., and Cholewicki, J., 2009. Changes in the mechanical properties of the trunk in low back pain may be associated with recurrence. J. Biomech. 42, 61-66.
Hoogendoorn, W. E., Bongers, P. M., de Vet, H. C. W., Douwes, M., Koes, B. W., Miedema, M. C., et al., 2000. Flexion and rotation of the trunk and lifting at work are risk factors for low back pain: Results of a prospective cohort study. Spine 25, 3087-3092.
Hwang, J. H., Lee, Y.-T., Park, D. S., and Kwon, T.-K., 2008. Age affects the latency of the erector spinae response to sudden loading. Clin. Biomech. 23, 23-29.
Kumar, S., 2001. Theories of musculoskeletal injury causation. Ergonomics 44, 17-47.
Larivière, C., Gravel, D., Gagnon, D., and Arsenault, A. B., 2009. Toward the development of predictive equations of back muscle capacity based on frequency- and temporal-domain electromyographic indices computed from intermittent static contractions. Spine J. 9, 87-95.
Lawrence, B. M., Buckner, G. D., and Mirka, G. A., 2006. An adaptive system identification model of the biomechanical response of the human trunk during sudden loading. J. Biomech. Eng. 128, 235-241.

McCook, D. T., Vicenzino, B., and Hodges, P. W., 2009. Activity of deep abdominal muscles increases during submaximal flexion and extension efforts but antagonist co-contraction remains unchanged. J. Electromyogr. Kinesiol. 19, 754-762.
McGill, S. M., and Brown, S., 1992. Creep response of the lumbar spine to prolonged full flexion. Clin. Biomech. 7, 43-46.
Moorhouse, K. M., and Granata, K. P., 2007. Role of reflex dynamics in spinal stability: Intrinsic muscle stiffness alone is insufficient for stability. J. Biomech. 40, 1058-1065.
Olson, M. W., Li, L., and Solomonow, M., 2004. Flexion-relaxation response to cyclic lumbar flexion. Clin. Biomech. 19, 769-776.
Punnett, L., and Wegman, D. H., 2004. Work-related musculoskeletal disorders: The epidemiologic evidence and the debate. J. Electromyogr. Kinesiol. 14, 13-23.
Radebold, A., Cholewicki, J., Panjabi, M. M., and Patel, T. C., 2000. Muscle response pattern to sudden trunk loading in healthy individuals and in patients with chronic low back pain. Spine 25, 947-954.
Reeves, N. P., and Cholewicki, J. P., 2010. Expanding our view of the spine system. Eur. Spine J. 19, 331-332.
Roffey, D. M., Wai, E. K., Bishop, P., Kwon, B. K., and Dagenais, S., 2010. Causal assessment of workplace manual handling or assisting patients and low back pain: Results of a systematic review. The Spine Journal 10, 639-651.
Rogers, E. L., and Granata, K. P., 2006. Disturbed paraspinal reflex following prolonged flexion-relaxation and recovery. Spine 31, 839-845.
Rosenbaum, D., and Hennig, E. M., 1995. The influence of stretching and warm-up exercises on achilles tendon reflex activity. J. Sports Sci. 13, 481 - 490.
Sanchez-Zuriaga, D., Adams, M. A., and Dolan, P., 2010. Is activation of the back muscles impaired by creep or muscle fatigue? Spine 35, 517-525.
Shin, G., and Mirka, G. A., 2007. An in vivo assessment of the low back response to prolonged flexion: Interplay between active and passive tissues. Clin. Biomech. 22, 965-971.
Solomonow, M., 2004. Ligaments: A source of work-related musculoskeletal disorders. J. Electromyogr. Kinesiol. 14, 49-60.
Solomonow, M., Baratta, R. V., Banks, A., Freudenberger, C., and Zhou, B. H., 2003a. Flexion-relaxation response to static lumbar flexion in males and females. Clin. Biomech. 18, 273-279.
Solomonow, M., Zhou, B. H., Baratta, R. V., Lu, Y., Zhu, M., and Harris, M., 2000. Biexponential recovery model of lumbar viscoelastic laxity and reflexive muscular activity after prolonged cyclic loading. Clin. Biomech. 15, 167-175.
Solomonow, M., Zhou, B. H., Baratta, R. V., and Burger, E., 2003b. Biomechanics and electromyography of a cumulative lumbar disorder: Response to static flexion. Clin. Biomech. 18, 890-898.
Solomonow, M., Zhou, B.-H., Baratta, R. V., Lu, Y., and Harris, M., 1999. Biomechanics of increased exposure to lumbar injury caused by cyclic loading: Part 1. Loss of reflexive muscular stabilization. Spine 24, 2426-2434.
Stokes, I. A. F., Fox, J. R., and Henry, S. M., 2006. Trunk muscular activation patterns and responses to transient force perturbation in persons with self-reported low back pain. Eur. Spine J. 15, 658-667.
Stokes, I. A. F., Gardner-Morse, M., Henry, S. M., and Badger, G. J., 2000. Decrease in trunk muscular response to perturbation with preactivation of lumbar spinal musculature. Spine 25, 1957-1964.
Toosizadeh, N., Bazrgari, B., Hendershot, B., Muslim, K., and Nussbaum, M. A., 2010. *In vivo* load-relaxation of the trunk with prolonged flexion*.* 34th Annual Meeting of the American Society of Biomechanics, Providence, Rhode Island. (http://www.asbweb.org/conferences/2010/abstracts/51.pdf)

Wai, E. K., Roffey, D. M., Bishop, P., Kwon, B. K., and Dagenais, S., 2010. Causal assessment of occupational bending or twisting and low back pain: Results of a systematic review. The Spine Journal 10, 76-88.
Zhang, L. -Q., Haiyun, H., Sliwa, J. A., and Rymer, W. Z., 1999. System identification of tendon reflex dynamics. Rehabilitation Engineering, IEEE Transactions on 7, 193-203.

5. Aim 3: Accumulation of Neuromuscular Disturbances from Repeated Static Flexion

Muslim K, Bazrgari B, Hendershot B, Toosizadeh N, Nussbaum MA, Madigan ML: [2012] Disturbance and recovery of trunk mechanical and neuromuscular behaviors following repeated static trunk flexion: influences of duration and duty cycle on creep-induced effect. Applied Ergonomics, Revision In Progress.

Abstract
Occupations involving frequent trunk flexion are associated with a higher incidence of low back pain. To investigate the effects of repeated static flexion on trunk behaviors, 12 participants completed six combinations of three static flexion durations (1, 2, and 4 min), and two flexion duty cycles (33% and 50%). Trunk mechanical and neuromuscular behaviors were obtained pre- and post-exposure and during recovery using sudden perturbations. A longer duration of static flexion and a higher duty cycle increased the magnitude of decrements in intrinsic stiffness. Increasing duty cycle caused larger decreases in reflexive muscle responses, and females had substantially larger decreases in reflexive responses following exposure. Patterns of recovery for intrinsic trunk stiffness and reflexive responses were consistent across conditions and genders, and none of these measures returned to pre-exposure values after 20 minutes of recovery. Reflexive responses may not provide a compensatory mechanism to offset decreases in intrinsic trunk stiffness following repetitive static trunk flexion. A prolonged recovery duration may lead to trunk instability and a higher risk of low back injury.

Introduction
Low back pain (LBP) remains the most prevalent musculoskeletal disorder around the world and involves a substantial economic burden (Baldwin, 2004; Dagenais et al., 2008; Jeffrey, 2006; Loney & Stratford, 1999; Luo et al., 2004; Manchikanti et al., 2009). An increased risk of LBP is associated with occupational tasks that involve repetitive lifting and prolonged trunk flexion (BLS, 2009; Hoogendoorn et al., 2000; Manchikanti, 2000; Marras, 2000). Although some disagreement remains regarding the level to which causality has been demonstrated (Kuijer et al., 2011; Wai et al., 2010), several studies have identified potential underlying mechanisms linking flexed working postures to the onset of LBP. Flexed postures can alter trunk passive mechanical properties and compromise active neuromuscular control of the spinal column as a consequence of decreased trunk proprioception (Gade & Wilson, 2007; Wilson & Granata, 2003). These alterations may adversely affect the mechanics of the spinal column, potentially leading to excessive spinal load and/or spinal instability, and increasing the risk for low back injury (Panjabi, 1992a, 1992b).

Recent studies indicate that a single period of exposure to static trunk flexion causes viscoelastic deformation of trunk soft tissues (e.g., muscles, discs, ligaments, and joint capsules) and alters trunk mechanical behaviors as indicated by reductions in intrinsic trunk stiffness (Bazrgari et al., 2011a; Hendershot et al., 2011; Little & Khalsa, 2005; McGill & Brown, 1992; Solomonow et al., 2003b). Reductions in intrinsic trunk stiffness require active neuromuscular compensation to maintain mechanical equilibrium and stability of the spine (Bazrgari et al., 2011a; Hendershot et al., 2011). However, static trunk flexion also alters the active neuromuscular behavior in that it reduces muscle force-generating capacity (Fowles et al., 2000; Weir et al., 2005), diminishes muscle spindle excitability (Avela et al., 1999; Solomonow, 2012), and may alter the ligament-muscle reflexive response (Le et al., 2009; Solomonow, 2009). Decreases in both intrinsic trunk stiffness and active neuromuscular behavior induced by trunk flexion may therefore increase the risk of developing LBP due to spinal instability.

Disturbances to trunk passive mechanical and active neuromuscular behaviors induced by trunk flexion can require a longer time for recovery than the initial exposure duration (Adams et al., 1990; Bazrgari et al., 2011a; Ekström et al., 1996; Hedman & Fernie, 1995; Hendershot et al., 2011; Keller et al., 1988; McGill & Brown, 1992). Static flexion that is repeated (e.g., during repetitive lifting as in agricultural and construction tasks) could therefore result in an accumulation of disturbances to trunk mechanical and neuromuscular behaviors due to incomplete recovery upon initiation of subsequent tasks. Hence, quantifying the acute changes in trunk passive mechanical and active neuromuscular behaviors following repeated trunk flexion is important for better understanding LBP etiology, and may aid in improving work design (e.g., work-rest cycles) in occupations involving frequent and/or repetitive flexed postures. The number of flexion cycles in task involving repetitive flexion can be characterized by the flexion duration and duty-cycle. Previous studies have confirmed that a longer duration of prolonged static flexion increases neuromuscular disturbances in the lumbar spine (Bazrgari et al., 2011a; Hendershot et al., 2011; LaBry et al., 2004). In studies using a feline model, short rest periods between flexion events have also been shown to have adverse effects (Courville et al., 2005; Sbriccoli et al., 2007). Therefore, the objective of the present study was to quantify the effects of flexion duration and duty cycle on trunk passive mechanical and active neuromuscular behaviors following repeated trunk flexion. We hypothesized that the severity of changes in trunk behaviors following repeated static flexion would increase with both flexion duration and duty cycle. We also expected that changes in passive mechanical behaviors following repeated flexion would not be adequately compensated by the active neuromuscular system, and that recovery would be prolonged and contingent on the severity of changes.

Methods

Participants

Twelve participants completed the study, and all were healthy adults with no self-reported history of low-back pain or current medical conditions that might have influenced the results. Participants included six males with mean (SD) age, stature, and body mass of 23.3 (1.9) yr, 177.9 (3.7) cm, and 71.6 (8.4) kg, respectively; corresponding values for the six females were 24.5 (2.3) yr, 162 (4.4) cm, and 56.7 (3.3) kg. A relatively young group of participants was included to avoid potential influences related to age. Prior to any data collection, each participant completed informed consent procedures approved by the Virginia Tech Institutional Review Board.

Experimental design and procedures

A repeated measures design was used, in which several measures of trunk mechanical and neuromuscular behaviors were obtained prior to and following exposures to repeated static trunk flexion. Participants completed six experimental sessions involving exposure to all combinations of three static flexion durations and two flexion duty cycles. Each session was conducted at a similar time on separate days, and with a minimum of two days between consecutive sessions. To reduce the potential for order-related confounding effects, the order of conditions was specified using balanced Latin Squares (i.e., two 6x6 squares, one for each gender group). During the experiment, participants stood in a rigid metal frame designed to restrain the pelvis and lower limbs in a fixed, but comfortable posture (Figure 1). Participants were then exposed to one of the six combinations of three flexion durations (1, 2, and 4 min) and two duty cycles (33% and 50%). This flexion-rest-flexion sequence was repeated continuously for 48 minutes, with the number of total cycles dependent on the flexion duration and duty cycle (Figure 2). Static flexion involved participants flexing their trunk forward as far as possible while relaxing their muscles, minimizing potential confounding effects of muscle fatigue.

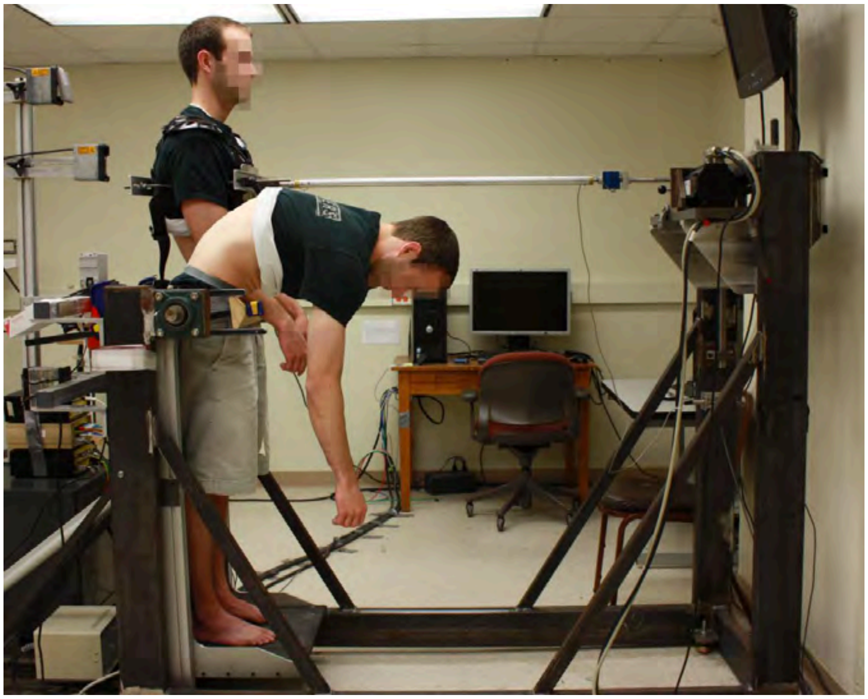

Figure 1: Experimental set-up demonstrating a participant during trunk mechanical and neuromuscular measurement, superimposed with a picture of the same participant in static flexion.

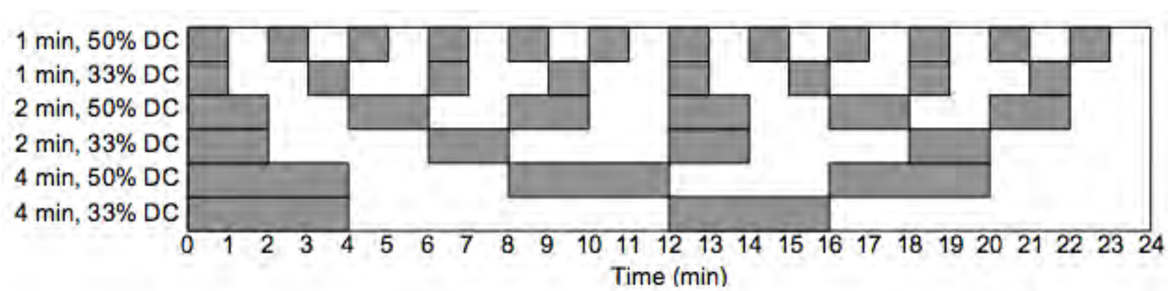

Figure 2: Illustration of the different conditions involving repeated static trunk flexion. Here, 24-min. periods are indicated, each of which was repeated two times in the experiment. Six conditions were tested, involving combinations of three exposure durations (1, 2, and 4 minutes) and two duty cycles (DC). Shaded areas indicate periods of flexion exposure.

Electromyograms (EMG) were recorded bilaterally using bipolar Ag/AgCl surface electrodes over the erector spinae at the L1 and L3 levels, external obliques, and rectus abdominis muscles (Bazrgari et al., 2011a; Hendershot et al., 2011). Prior to applying electrodes, the skin was prepared using abrasion and cleaned with alcohol to achieve an inter-electrode impedance below 10 KΩ. Raw EMG signals were pre-amplified (x100) near the collection site, bandpass filtered (10-500Hz), amplified (x100), and converted to root mean square (RMS) in hardware (Measurement Systems Inc., Ann Arbor, MI, USA), then sampled at 1000 Hz. Trunk posture was monitored using a 6 DOF inertial measurement unit (IMU: Xsens, Los Angeles, CA, USA) affixed to the trunk dorsum at the T12 level and sampled at 100 Hz. As described below, trunk mechanical behaviors (i.e. intrinsic trunk stiffness and trunk apparent mass) and neuromuscular behaviors (i.e. reflex gain, reflex delay, maximum reflex force, and timing of maximum reflex force) were recorded in an upright posture prior to and immediately following the 48-minute exposures. These measurements were also obtained after 2.5, 5, 10 and 20 minutes of recovery post exposure, again in the upright posture. Prior to a period of static flexion, there was a ~10 second delay resulting from the need to remove the measurement equipment and assume a flexed posture. Subsequent to a flexion period, roughly 45 seconds were required for the participant to stand upright, to reattach the measurement equipment, and to initiate the mechanical and neuromuscular measurements.

As in our previous work (Bazrgari et al., 2011a; Hendershot et al., 2011), a pseudorandomly-timed sequence of 12 anterior-posterior position perturbations of ±5 mm were applied to the trunk at ~T8 via a servomotor (Kollmorgen, Radford, VA), rigid rod, and chest harness. Participants maintained a consistent sub-maximal extensor effort (or "preload") using real-time visual feedback of RMS EMG of the bilateral L3 erector spinae, for ~3 sec prior to and during the perturbation sequences. The target effort was set to 10% of maximal EMG, the latter determined from maximum voluntary exertion efforts completed at the beginning of each experimental session. The use of extensor EMG as visual feedback for controlling the preload was expected to minimize potential influences of alterations in muscle recruitment (e.g. changes in co-contraction). Trunk horizontal displacements at the T8 level were measured using both the servomotor encoder and a laser displacement sensor (Keyence, Osaka, Japan), while reaction forces were measured using an in-line load cell (Interface SM2000, Scottsdale, AZ, USA). These data were sampled at 1000 Hz, then low-pass filtered using a 7th-order, bidirectional, Butterworth filter with a 10 Hz cutoff frequency (Bazrgari et al., 2011b).

Trunk Mechanical and Neuromuscular Measures

Creep deformation was assessed by changes in trunk full flexion angle (measured by the IMU) over the 48-minute exposures. Specifically, accumulated creep was defined as the change in full flexion angle from the initial to the final flexion periods. Reflex delay was determined as the time between perturbation onset and reflexive muscle response for each anteriorly-directed perturbation (via EMG); the former was determined when the absolute value of measured trunk velocity (from the laser sensor) exceeded zero and the latter identified when the measured erector spinae EMG exceeded two standard deviations above mean activity prior to the perturbations (Granata et al., 2004; Hendershot et al., 2011; Zhang et al., 1999). Trunk mechanical behaviors were quantified by relating measured trunk kinematics to trunk kinetics during the time period from perturbation onset to reflex onset (Bazrgari et al., 2011b; Hendershot et al., 2011). Parameters of a model representing the trunk (apparent mass and intrinsic trunk stiffness) were estimated using a least-squares curve fit in MATLAB™ (MathWorks, Natick, MA, USA); this was done separately for each anteriorly-directly perturbation.

To characterize trunk neuromuscular behaviors, reflex forces were first estimated by subtracting the model-estimated intrinsic force contribution from the total measured trunk response (i.e., trunk reaction force measured by the inline load cell). Magnitude and timing (with respect to perturbation onset) of the maximum reflex force were quantified to represent the overall trunk reflexive behavior (Bazrgari et al., 2011a); this analysis was limited to a time window of 150 ms following reflex onset to avoid voluntary responses. The instantaneous reflex force during this same time window was also correlated to time-shifted (equal to reflex delay) trunk velocity to estimate reflex gain (Moorhouse & Granata, 2007). Reflex gain is a measure of the magnitude of the reflex response with respect to trunk velocity, and a larger gain indicates a greater reflex response. Such reflex measures represent a more global measure of trunk reflexive behavior, differing from EMG-driven estimates that typically rely on measured EMG responses of superficial trunk muscles.

Dependent measures were thus: creep deformation, apparent mass, intrinsic stiffness, reflex gain, maximum reflex force, reflex delay, and the timing of maximum reflex forces. Pre-exposure differences in these trunk behaviors between genders were evaluated using analyses of variance (ANOVA). Post-exposure measures were normalized to pre-exposure values [(post-pre)/pre]. Effects of flexion duration, duty cycle, and gender on accumulated creep and measures of trunk behavior were then assessed using separate mixed-factor ANOVAs for each measure. No substantial deviations from parametric model assumptions were evident. For dependent measures that exhibited post-exposure effects of duration, duty cycle, or gender, MANOVAs were used to assess the effects of these same factors over the 20-minute recovery periods. Where sphericity violations were found in MANOVA (for reflexive measures), the Geisser-Greenhouse correction was used. As relevant, post-hoc pairwise comparisons were performed using Tukey's Honestly Significant Difference (HSD) test, and significant interaction effects were explored using simple effects analyses. Contrasts, using a Scheffé correction, were used to explore the effects of cycle time on the post-exposure measures, in sets of conditions having the same total flexion duration (see Figure 2). Two specific sets of contrasts were made between: 1) three conditions involving 0.5 min of flexion per minute (1, 2, and 4 min. exposures with 50% duty cycle); and 2) three conditions involving 0.33 min of flexion per minute (1, 2, and 4 min. exposures with 33% duty cycle). Creep deformation data from one trial (male, 4 min. duration, 50% duty cycle) was excluded from analysis due to measurement error; other data from this trail were unaffected. All statistical analyses were done using JMP™ (Version 9, SAS Institute Inc., Cary, NC), and significance was determined when $P < .05$. Summary values are reported as means (SD).

Results

Pre-exposure

Both estimated apparent trunk mass and intrinsic trunk stiffness were larger among males than females (Table 1). Reflex delays were comparable between genders, as were reflex gains and the magnitude and timing of maximum reflex forces.

Table 1. Pre-exposure measures of trunk behavior. Mean (SD) values are shown for each gender, and significant differences between genders are indicated by bolded *P*-values.

Measure	Males	Females	$F_{(1,10)}$	*P*-value
Apparent Mass (kg)	21 (3.6)	17.2 (2.5)	5.89	**0.036**
Intrinsic Stiffness (N/m)	8300 (1262)	6313 (901)	14.71	**0.0033**
Reflex Gain (Ns/m)	1133 (302)	1130 (276)	0.0004	0.98
Reflex Delay (ms)	61.9 (5.2)	63.9 (3.6)	1.16	0.31
Max. Reflex Force (N)	205 (52.4)	202.4 (45.1)	0.015	0.91
Timing of Max. Reflex Force (ms)	154.8 (8.5)	157.3 (9.6)	0.3	0.59

Post-exposure

Accumulated creep deformation across all conditions was 12.3 (7.8)°, and was not different ($F_{(1,10)}$=0.56; P=0.47) between females [11.6 (8.7)°] and males [13.2 (6.8)°]. Increases in flexion duration significantly increased creep deformation ($F_{(2,44)}$ = 3.4; P=0.042), with values of 10.2 (7.5)°, 11.8 (7.5)°, and 15.0 (7.8)° following repeated flexion of 1, 2, and 4 minutes, respectively (Figure 3). Creep deformation also increased with increasing duty cycle, an effect that approached significance ($F_{(1,44)}$ = 3.86; P=0.056), with values of 10.8 (7.6)° and 13.8 (7.8)° in the 33% and 50% duty cycle conditions, respectively. There was a significant interaction between duty cycle and gender ($F_{(1,44)}$ = 4.29; P=0.044). Specifically, a larger duty cycle increased (P = 0.006) creep deformation among females, with respective values of 8.3 (7.8)° and 14.6 (8.6)° in the 33% and 50% duty cycle, whereas these were unchanged among males (P = 0.94). Though the interaction effect only approached significance ($F_{(2,44)}$ = 3.08; P=0.056), the effects of duration on creep deformation also differed between genders. Creep deformation increased with flexion duration among males, with respective values of 8.4 (4.5)°, 13.6 (6.3)°, 17.9 (6.3)° for the 1, 2, and 4 minutes exposures, but it had relatively little influence among females.

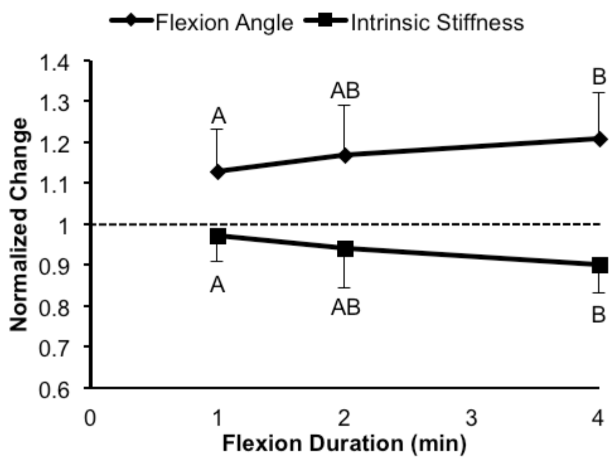

Figure 3: Effects of flexion duration on flexion angle (indicating creep deformation) and intrinsic trunk stiffness. Normalized changes are illustrated (e.g., 1.1 indicates a 10% increase from pre-exposure values). Error bars indicate standard deviations, and results from pairwise comparisons (within each measure) are indicated by letters.

Following 48 minutes of repeated flexion, there were significant changes in all other dependent measures (Table 2), each of which decreased following exposure. For some measures, these changes were modified by main effects of duration, duty cycle, or gender as well as by some interactive effects. The decrease in intrinsic trunk stiffness following exposure was larger with increasing duration (Figure 3), and a larger decrease was also found in the 50% vs. 33% duty cycles (Figure 4). Flexion duration had different effects on intrinsic stiffness between genders ($F_{(2,45)}$ = 4.18; P=0.022); both genders had similar responses to the 1- and 4-minute exposures, though males had smaller effects following the 2-minute exposures.

Duty cycle and gender, though not duration, significantly influenced reflex gain and maximum reflex force. Both reflex measures decreased more substantially following exposures to the 50% duty cycle (Figure 4). Reflex gain decreased by 13.3 (17.1) and 2.4 (20.7)% among females and males, respectively, whereas respective decreases in maximum reflex forces were 11.4 (11.8) and 0.3 (14.4) %. Decreases in apparent mass, reflex delay, and timing of maximum reflex force were not influenced by the independent variables. Muscle activity (total across all muscles) during the ~3 s prior to perturbations was comparable pre- versus post-exposure across all conditions and genders ($t_{(71)}$ = -0.72, P = 0.47), with respective values of 0.054 (0.009) and 0.053 (0.01) mV.

Table 2. Measures of trunk mechanical and neuromuscular behaviors obtained pre- and post-exposure to trunk flexion (significant main effects indicated by shaded cells). Means (SDs) are indicated.

Variable	Value		Change (Post–Pre)	Main effect		
	Pre	Post		Duration	Duty Cycle	Gender
Apparent Mass (kg)	19.1 (3.6)	18.7 (3.7)	$t_{(71)}$ = -3.39, P=0.0011	$F_{(2,45)}$ = 0.17, P=0.84	$F_{(1,45)}$=3.68, P=0.061	$F_{(1,10)}$ = 1.1, P=0.32
Intrinsic Stiffness (N/m)	7306 (1479)	6825 (1365)	$t_{(71)}$ = -6.61, P=0.0001	$F_{(2,45)}$ = 6.6, P=0.0031	$F_{(1,45)}$=5.95, P=0.019	$F_{(1,10)}$ =1.31, P=0.28
Reflex Gain (Ns/m)	1132 (287)	1060 (317)	$t_{(71)}$ = -2.7, P=0.0086	$F_{(2,45)}$= 0.21, P=0.81	$F_{(1,45)}$ = 5.17, P=0.028	$F_{(1,10)}$ =18.77, P=0.0015
Max. Reflex Force (N)	203.7 (48.5)	191.3 (51.2)	$t_{(71)}$ = -3.61, P=0.0006	$F_{(2,45)}$=0.077, P=0.93	$F_{(1,45)}$ = 8.36, P=0.0059	$F_{(1,10)}$ =9.51, P=0.015
Reflex delay (ms)	62.9 (4.6)	61.3 (4)	$t_{(71)}$ = -2.74, P=0.016	$F_{(2,45)}$= 1.2, P=0.31	$F_{(1,45)}$ = 0.77, P=0.39	$F_{(1,10)}$ = 0.37, P=0.56
Timing of Max. Reflex Force (ms)	156.1 (9.1)	153.3 (10.9)	$t_{(71)}$ = -4.64, P=0.0001	$F_{(2,45)}$ = 0.38, P=0.69	$F_{(1,45)}$ = 0.47, P=0.5	$F_{(1,10)}$= 2.44, P=0.15

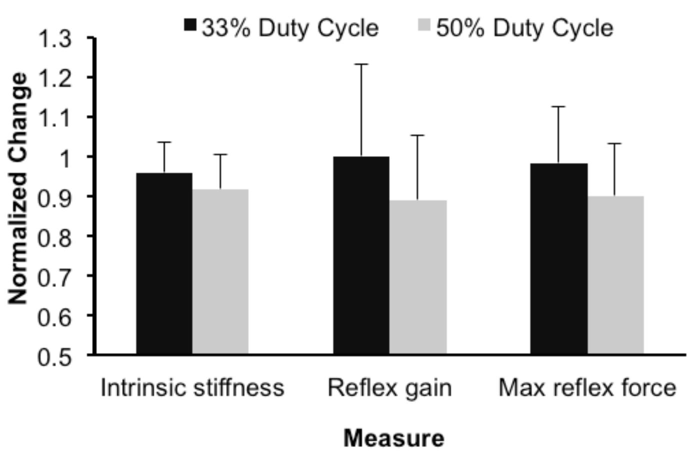

Figure 4: Effects of duty cycle on intrinsic stiffness, reflex gain, and maximum reflex force. Normalized changes are illustrated (e.g., 0.9 indicates a 10% decrease from pre-exposure values). Error bars indicate standard deviations. Differences between duty cycles were significant for all three measures.

Cycle time had significant effects only on intrinsic stiffness, for which shorter cycle times had smaller effects. With a 50% duty cycle, use of 1 min. exposure periods vs. 2 and 4 min. as a group led to significantly smaller decreases in intrinsic stiffness (-2.8% vs. -10.8%, respectively). In the case of a 33% duty cycle, the shorter exposure durations (1 and 2 min.) as a group also led to significantly smaller decreases in intrinsic stiffness (-2.6% vs. -7.6%, respectively).

Recovery

Following 20 minutes of standing recovery, and regardless of duty cycle and flexion duration, neither intrinsic trunk stiffness ($t_{(70)}$ = -4.64, P = 0.0001), reflex gain ($t_{(68)}$ = -3.71, P = 0.0004), or maximum reflex force ($t_{(69)}$ = -2.75, P = 0.0075) recovered to pre-exposure values. In contrast, apparent mass ($t_{(70)}$ = -0.081, P = 0.94) and reflex delay ($t_{(69)}$ = -1.04, P = 0.3) recovered at the first recovery measurements (i.e., 2.5 min after exposure). The timing of maximum reflex force remained consistently higher than pre-exposure values throughout the recovery period. No significant interaction effects between the independent variables and time were found (P > 0.12), and no qualitative differences were evident (Figures 5 and 6), indicating generally consistent patterns of stiffness recovery over time following all exposure conditions.

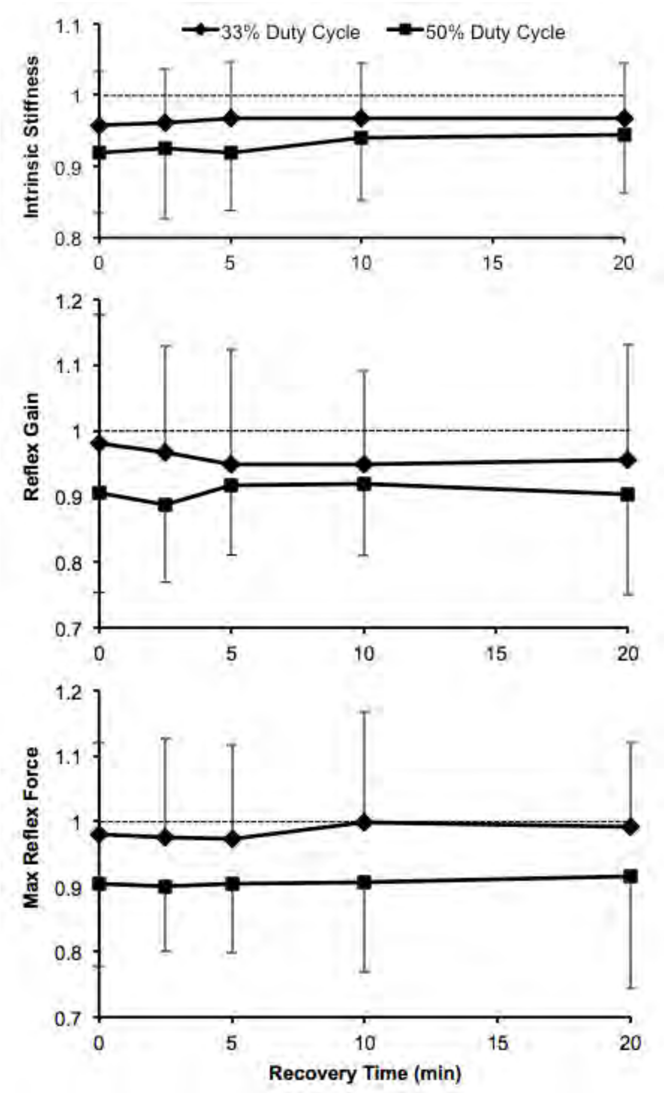

Figure 5: Recovery of normalized intrinsic trunk stiffness and reflex gain, for the two duty cycles, following 48-minute of repeated static flexion exposure. Error bars indicate standard deviations, and time = 0 indicates the end of the exposure period.

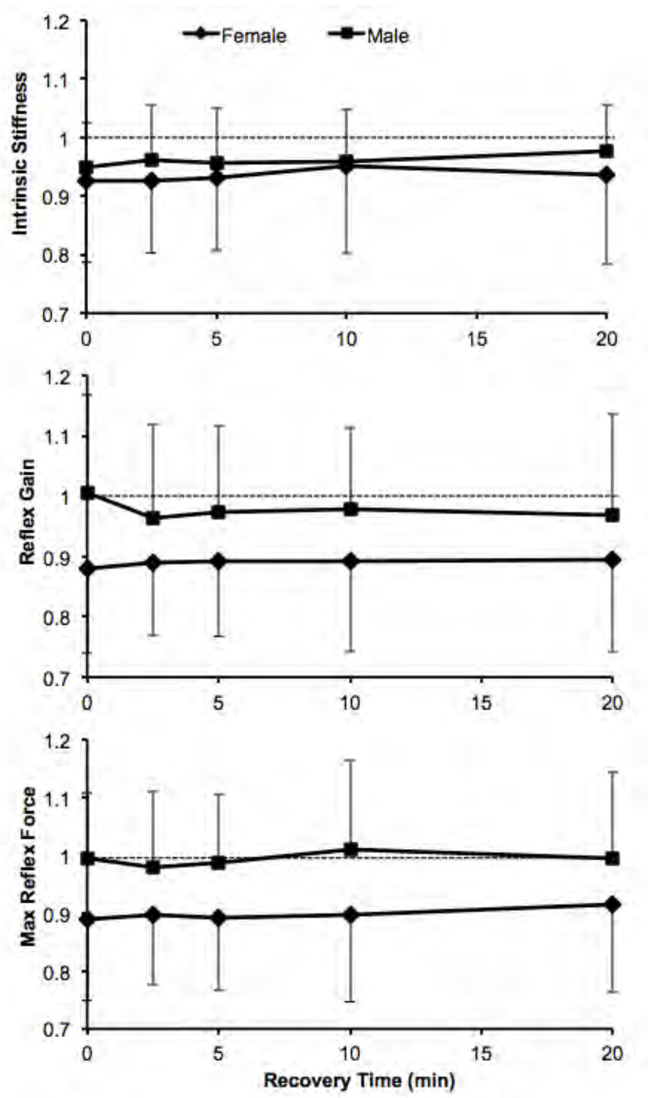

Figure 6: Recovery of normalized intrinsic stiffness, reflex gain, and reflex magnitude for both genders. Error bars indicate standard deviations, and time = 0 indicates the end of the exposure period.

Discussion

Creep deformation

Creep deformation in the present study indicates substantial alterations in trunk viscoelastic tissues following repeated static flexion. Across all conditions, the magnitude of accumulated creep [12.4 (7.8)°] was greater than the total creep following a single 10-min exposure to prolonged flexion [9.4 (5.2)°] reported earlier (Bazrgari et al., 2011a), which suggests a gradual accumulation over repeated exposures to static flexion as hypothesized. Existing mechanical/histological literature indicates that cyclic loads applied to ligaments result in altered mechanical properties (Carpenter et al., 1998; Safran, 1995; Soslowsky et al., 2000) and causes intervertebral discs to resist an increased proportion of the bending moment applied to the osteo-ligamentous spine (Adams & Dolan, 1996), which implies ligaments resisted a decreased proportion of the bending moment. Among parameters characterizing repetitive flexion, flexion duration was found here to influence creep deformation more substantially than duty cycle; specifically, longer flexion durations resulted in a larger creep deformation (Figure 3). Males had larger creep in response to longer flexion durations, and this was probably caused by the larger mass that loaded the spine during exposure.

Intrinsic trunk behavior

Intrinsic trunk stiffness decreased with flexion exposure, and this decrease was more substantial with both increasing flexion duration and duty cycle. In parallel with creep deformation (Figure 3), rest periods between flexion exposures did not have been sufficient to allow for full recovery, thus inducing an accumulation of alterations in intrinsic trunk stiffness over time. Since muscle activity during measurements was kept at a roughly constant level, the observed decrement in intrinsic trunk stiffness was likely caused by changes in passive mechanical trunk properties following repeated flexion exposures, and not by changes in background muscle activity (Cholewicki et al., 2000; Gardner-Morse & Stokes, 2001; Miller et al., 2010).

The overall decrease in intrinsic trunk stiffness, of approximately 3-10% across all treatments, is comparable to our earlier study with a single prolonged flexion exposure involving soft tissue creep (Bazrgari et al., 2011a), but is less than was observed following stress relaxation (Hendershot et al., 2011). In the latter study, larger relative decreases (i.e., 10-20%) were likely due to differences in experimental protocols (i.e., stress relaxation vs. creep deformation) and potentially to changes in harness design. A newer harness design used here connected the thorax to the perturbing device more tightly; it has demonstrated improved reliability (Hendershot et al., In press) and is believed to have provided more precise estimates of changes in intrinsic stiffness. Otherwise, absolute decreases in intrinsic stiffness across all conditions in the present study (i.e. 481 (617) N/m) are comparable in magnitude to our earlier results (i.e. 372 (824) N/m).

Intrinsic trunk stiffness recovered slowly after flexion exposure, as observed earlier (Bazrgari et al., 2011a; Hendershot et al., 2011). While intrinsic trunk stiffness remained at consistently lower values during recovery in the 50% duty cycle, full recovery was not observed in any of the exposure conditions following 20 minutes of standing rest. To reduce the risk of lumbar disorders, earlier studies have recommended providing a sufficient rest period of at least the same as, or longer than, the exposure duration in a repetitive flexion task (Hoops et al., 2007; Sbriccoli et al., 2007). A duty cycle of 33% here theoretically provided this recommended rest period between each flexion. However, even after completing 48 minutes of repeated static flexion with a duty cycle of 33%, and a 20-minute standing rest period following exposure, changes in trunk stiffness (and reflexive behaviors, as noted below) still were not found be fully recovered. As such, this recommended duration may not be sufficient to provide complete recovery of trunk behaviors, and other investigations using a feline model have reported that disturbances to viscoelastic tissues following cyclic work may last for several hours (Solomonow, 2012; Solomonow et al., 2003b).

Reflexive trunk behavior

Reflex gain and maximum reflex force, which assess the magnitude (vs. timing) of reflexive responses, decreased in all conditions following repeated static flexion, but more substantially with increasing duty cycle (Figure 4). This indicates a potential difference in the effects of repeated vs. prolonged flexion tasks, the latter of which has been found to increase reflexive responses in several studies (Bazrgari et al., 2011a; Granata et al., 2005; Hendershot et al., 2011). In these previous studies, it was argued that the increase in reflexive response is an immediate hyper-excitability as a compensatory mechanism in response to decreased intrinsic trunk stiffness. Such a mechanism was not evident here following repeated static flexion, which might be a result of more severe changes in viscoelastic tissues that either delayed muscle hyper-excitability or completely suppressed it. Consistent with this, earlier studies with a feline model have reported a decrease in EMG reflexive responses following cyclic flexion (Solomonow, 2012; Solomonow et al., 2003a; Solomonow et al., 1999). In addition, a study on human participants by Rogers & Granata (2006) reported similar behavior following prolonged trunk flexion tasks with intermittent rest periods. Repeated passive stretching of skeletal muscles surrounding other joints in the human body has also resulted in a similar decrease in reflexive responses (Avela et al., 1999; Rosenbaum & Hennig, 1995). This decrease in reflexive responses may be related to the increase in creep deformation with a higher duty cycle found here. Laxity/creep in viscoelastic tissues may also have contributed to decrements in the sensitivity of mechanoreceptors, increasing muscle afferent thresholds and causing a decrease in the activation of motor units (Solomonow, 2012). A decreased reflexive response in addition to reduced stiffness suggests, furthermore, that repeated static flexion may induce spinal instability, and which in turn may lead to spine injury and LBP (Panjabi, 1992a).

Similar to intrinsic stiffness, reflex gain and maximum reflex force also failed to return to pre-exposure values during 20 minutes of standing recovery, and the present results confirm earlier work that the recovery of reflexive responses is dependent upon the history of trunk loading (Bazrgari et al., 2011a; Hendershot et al., 2011). Our earlier studies suggest an increase in reflex gain and maximum reflex force following prolonged flexion, and this was suggested as a mechanism compensating for the yet-unrestored reductions in intrinsic stiffness (Bazrgari et al., 2011a; Hendershot et al., 2011). In Hendershot et al. (2011), increased reflex gain continued even after complete recovery of intrinsic trunk stiffness, while in Bazrgari et al. (2011a) increases in the reflex gain and maximum reflex force did not appear to persist sufficiently to compensate for decreases in intrinsic trunk stiffness during the recovery period. This discrepancy is arguably attributed to a difference in loading history in the two studies: the former used a stress relaxation protocol that has substantially lower loading (i.e., no gravitational moment from the upper body mass) compared to creep deformation induced by static flexion in the latter study. Therefore, a greater loading history, such as repeated exposure to static flexion that induces more creep deformation presented in this study, results in a slower recovery. Further, during recovery both intrinsic trunk stiffness and reflexive responses are simultaneously diminished, perhaps indicating an increased vulnerability to injury. Residual creep deformation during the recovery period may still be present, which as was noted earlier could influence the threshold of mechanoreceptors initiating reflexive responses. Unfortunately, residual creep angles were not collected during the recovery period, a limitation of the present study. Nevertheless, investigations using a feline model as reviewed by Solomonow (2012) suggest that reduced EMG reflexive responses persist for several hours following cyclic flexion exposure. Since the recovery period here was only 20 minutes, only the reduced reflexive response phase was likely captured, and not the complete recovery. In general, however, the present results are consistent with existing evidence in that the recovery process for reflexive responses may be slow.

Studies using a feline model have also reported a phenomenon of muscle hyper-excitability during the early recovery period following cyclic flexion (Hoops et al., 2007; Sbriccoli et al., 2007; Solomonow, 2012; Solomonow et al., 2003a). This EMG-based reflexive measure indicates increased muscle force as a compensatory response to increased creep deformation and microdamage of viscoelastic tissues in the lumbar spine (McLain & Pickar, 1998; Sekine et al., 2001; Solomonow, 2009). Hyper-excitability may improve trunk stiffness and serve to protect against injury (Solomonow, 2012). Solomonow (2012) suggests that initial hyper-excitability occurs within an hour following 120 minutes of cyclic flexion exposure, and Hoops et al. (2007) reported initiation during the first 10 minutes of recovery. Here, small initial muscle hyper-excitability was indicated by increased reflex gain, appearing in the first 5-minutes of recovery (for the case of 50% duty cycle; Fig. 4). Interestingly, reflex gain continued to decrease in the first 2.5-minutes of the recovery period, indicating potential residual effects following the exposure. However, in the 33% duty cycle condition, there was only a pattern of continued decrease and slow recovery of the reflexive response without any apparent muscle hyper-excitability. It is important to note, though, that the smallest reflex gain in the 33% duty cycle during the first 10 minutes of recovery was still larger than the biggest reflex gain in the 50% duty cycle. This pattern might be caused by a longer rest period between flexion inherent to the 33% duty cycle, inducing less of a disturbance to the neuromuscular system compared to the 50% duty cycle.

In most conditions, and especially for the 50% duty cycle and longer flexion durations, females had substantially lower reflexive responses (both reflex gain and max reflex force, Figure 6). Combined with a lower intrinsic trunk stiffness, as found here and elsewhere (Bazrgari et al., 2011a; Hendershot et al., 2011), this may expose females to decreased control of spinal stability following repeated static flexion. This gender difference is also notably consistent with the higher incidence rate of LBP among females (Pleis et al., 2010), and offers a potential underlying biomechanical/physiological mechanism.

Implications of results

Occupations involving prolonged or frequent flexion tasks are associated with higher risks of LBP (Hoogendoorn et al., 2000; Manchikanti, 2000). These risks may be correlated with, or perhaps even caused by (at least in part), disturbances in trunk behaviors caused by flexion exposure. Our current and recent findings, along with work by others, suggest that both prolonged and repeated static trunk flexion results in creep deformation of trunk viscoelastic tissues in humans. As a result of this deformation, intrinsic trunk stiffness substantially decreases following both prolonged and repeated static flexion. However, an immediate increase in reflexive response observed earlier following prolonged flexion was not evident at the end of the present repeated static flexion tasks; instead, reflexive responses were decreased following cyclic flexion. This decrease in reflexive response, along with a concurrent decrease in intrinsic trunk stiffness, may reduce spinal stability and increase the risk of mechanical injury to the spine. In addition, recovery of both intrinsic trunk stiffness and reflexive responses may be slow. A compensatory reflexive response from the neuromuscular system, in response to decreases in intrinsic trunk stiffness, was either absent or of insufficient magnitude during 20 minute of recovery. Such a situation suggests a vulnerable state even after the completion of repeated static flexion. Regardless of the duration, a longer rest period between consecutive flexions (i.e., lower duty cycle) induces less accumulated creep deformation, and smaller subsequent decreases in intrinsic trunk stiffness and reflexive responses. As such, performing repeated flexion tasks at a lower duty cycle may be beneficial for reducing the risk of low back injury. The observed effects of cycle time may also have potential relevance to job design. Comparisons of cases wherein the total flexion exposure duration was consistent, indicated that shorter cycle times led to less substantial effects on intrinsic stiffness. However, more frequent transitions between flexion and recovery could impose additional musculoskeletal demands (i.e., muscle and spinal loads involved in changing trunk postures). Cycle time is also an important aspect to the use of job rotation, a common administrative control (Jorgensen et al., 2005). If the current results generalize to longer cycle times, it may be beneficial to adopt more frequent rotation between tasks involving (i.e., that have varying levels of trunk flexion exposures). Further study is clearly needed to confirm the potential benefits of the noted design approaches, as well as to examine a wide range of trunk flexion exposures and in a wider and more representative range of participants.

Acknowledgments

This work was supported by award R01-OH008504 from the Centers for Disease Control and Prevention (CDC). Its contents are solely the responsibility of the authors and do not necessarily represent the official views of the CDC. The authors thank Mr. Waldron and Mr. Vest for their assembly and maintenance of the testing apparatus. The authors dedicate this work to Dr. Kevin P. Granata, whose original ideas formed the foundation for the current investigation.

References

Adams, M., & Dolan, P., 1996. Time-dependent changes in the lumbar spine's resistancc to bending. Clinical Biomechanics, 11(4), 194-200.

Adams, M., Dolan, P., Hutton, W., & Porter, R., 1990. Diurnal changes in spinal mechanics and their clinical significance. Journal of Bone and Joint Surgery-British Volume, 72(2), 266.

Avela, J., Kyröläinen, H., & Komi, P. V., 1999. Altered reflex sensitivity after repeated and prolonged passive muscle stretching. Journal of Applied Physiology, 86(4), 1283.

Baldwin, M. L., 2004. Reducing the costs of work-related musculoskeletal disorders: targeting strategies to chronic disability cases. Journal of Electromyography and Kinesiology, 14(1), 33-41.

Bazrgari, B., Hendershot, B., Muslim, K., Toosizadeh, N., Nussbaum, M. A., & Madigan, M. L., 2011a. Disturbance and recovery of trunk mechanical and neuromuscular behaviours following prolonged trunk flexion: influences of duration and external load on creep-induced effects. Ergonomics, 54(11), 1043-1052.

Bazrgari, B., Nussbaum, M. A., & Madigan, M. L., 2011b. Estimation of trunk mechanical properties using system identification: effects of experimental setup and modelling assumptions.

BLS, 2009. Nonfatal occupational injuries and illnesses requiring days away from work: Washington, DC: Bureau of Labor Statics, US Department of Labor.

Carpenter, J. E., Flanagan, C. L., Thomopoulos, S., Yian, E. H., & Soslowsky, L. J., 1998. The effects of overuse combined with intrinsic or extrinsic alterations in an animal model of rotator cuff tendinosis. The American journal of sports medicine, 26(6), 801.

Cholewicki, J., Simons, A. P. D., & Radebold, A., 2000. Effects of external trunk loads on lumbar spine stability. Journal of biomechanics, 33(11), 1377-1385.

Courville, A., Sbriccoli, P., Zhou, B. H., Solomonow, M., Lu, Y., & Burger, E. L., 2005. Short rest periods after static lumbar flexion are a risk factor for cumulative low back disorder. Journal of Electromyography and Kinesiology, 15(1), 37-52.

Dagenais, S., Caro, J., & Haldeman, S., 2008. A systematic review of low back pain cost of illness studies in the United States and internationally. The Spine Journal, 8(1), 8-20.

Ekström, L., Kaigle, A., Hult, E., Holm, S., Rostedt, M., & Hansson, T., 1996. Intervertebral disc response to cyclic loading - an animal model. Proceedings of the Institution of Mechanical Engineers, Part H: Journal of Engineering in Medicine 1989-1996 (vols 203-210), 210(48), 249-258.

Fowles, J., Sale, D., & MacDougall, J., 2000. Reduced strength after passive stretch of the human plantarflexors. Journal of Applied Physiology, 89(3), 1179.

Gade, V. K., & Wilson, S. E., 2007. Position sense in the lumbar spine with torso flexion and loading. Journal of applied biomechanics, 23(2), 93.

Gardner-Morse, M. G., & Stokes, I. A. F., 2001. Trunk stiffness increases with steady-state effort. Journal of biomechanics, 34(4), 457-463.

Granata, K. P., Rogers, E., & Moorhouse, K., 2005. Effects of static flexion-relaxation on paraspinal reflex behavior. Clinical Biomechanics, 20(1), 16-24.

Granata, K. P., Slota, G., & Bennett, B., 2004. Paraspinal muscle reflex dynamics. Journal of biomechanics, 37(2), 241-247.
Hedman, T. P., & Fernie, G. R., 1995. In vivo measurement of lumbar spinal creep in two seated postures using magnetic resonance imaging. Spine, 20(2), 178.
Hendershot, B., Bazrgari, B., Muslim, K., Toosizadeh, N., Nussbaum, M. A., & Madigan, M. L., 2011. Disturbance and recovery of trunk stiffness and reflexive muscle responses following prolonged trunk flexion: Influences of flexion angle and duration. Clinical Biomechanics, 26(3), 250-256.
Hendershot, B., Bazrgari, B., Nussbaum, M. A., & Madigan, M. L., In press. Within- and between-day reliability of trunk mechanical behaviors estimated using position-controlled perturbations. Journal of Biomechanics.
Hoogendoorn, W. E., Bongers, P. M., de Vet, H. C. W., Douwes, M., Koes, B. W., Miedema, M. C., et al., 2000. Flexion and rotation of the trunk and lifting at work are risk factors for low back pain: results of a prospective cohort study. Spine, 25(23), 3087.
Hoops, H., Zhou, B. H., Lu, Y., Solomonow, M., & Patel, V., 2007. Short rest between cyclic flexion periods is a risk factor for a lumbar disorder. Clinical Biomechanics, 22(7), 745-757.
Jeffrey, N., 2006. Lumbar disc disorders and low-back pain: socioeconomic factors and consequences. The Journal of Bone and Joint Surgery (American), 88(suppl_2), 21-24.
Jorgensen, M., Davis, K., Kotowski, S., Aedla, P., & Dunning, K., 2005. Characteristics of job rotation in the Midwest US manufacturing sector. Ergonomics, 48(15), 1721-1733.
Keller, T., Hansson, T., Holm, S., Pope, M., & Spengler, D., 1988. In vivo creep behavior of the normal and degenerated porcine intervertebral disk: a preliminary report. Journal of spinal disorders, 1(4), 267.
Kuijer, P., Frings-Dresen, M. H. W., Gouttebarge, V., van Die√´n, J. H., van der Beek, A. J., & Burdorf, A., 2011. Low back pain: we cannot afford ignoring work. The Spine Journal, 11(2), 164-164.
LaBry, R., Sbriccoli, P., Zhou, B. H., & Solomonow, M., 2004. Longer static flexion duration elicits a neuromuscular disorder in the lumbar spine. Journal of Applied Physiology, 96(5), 2005-2015.
Le, B., Davidson, B., Solomonow, D., Zhou, B. H., Lu, Y., Patel, V., et al., 2009. Neuromuscular control of lumbar instability following static work of various loads. Muscle & Nerve, 39(1), 71-82.
Little, J. S., & Khalsa, P. S., 2005. Human lumbar spine creep during cyclic and static flexion: creep rate, biomechanics, and facet joint capsule strain. Annals of biomedical engineering, 33(3), 391-401.
Loney, P., & Stratford, P., 1999. The prevalence of low back pain in adults: a methodological review of the literature. Physical therapy, 79(4), 384.
Luo, X., Pietrobon, R., X Sun, S., Liu, G. G., & Hey, L., 2004. Estimates and patterns of direct health care expenditures among individuals with back pain in the United States. Spine, 29(1), 79.
Manchikanti, L., 2000. Epidemiology of low back pain. Pain Physician, 3(2), 167-192.
Manchikanti, L., Singh, V., Datta, S., Cohen, S., & Hirsch, J., 2009. Comprehensive review of epidemiology, scope, and impact of spinal pain. Pain Physician, 12(4), E35.
Marras, W., 2000. Occupational low back disorder causation and control. Ergonomics, 43(7), 880-902.
McGill, S., & Brown, S., 1992. Creep response of the lumbar spine to prolonged full flexion. Clinical Biomechanics, 7(1), 43-46.
McLain, R. F., & Pickar, J. G., 1998. Mechanoreceptor endings in human thoracic and lumbar facet joints. Spine, 23(2), 168.
Miller, E., Bazrgari, B., Hendershot, B., Nussbaum, M., & Madigan, M., 2010. Dynamic response of the trunk to position perturbations,Äìeffects of gender, preload, and trunk angle.

Moorhouse, K. M., & Granata, K. P., 2007. Role of reflex dynamics in spinal stability: intrinsic muscle stiffness alone is insufficient for stability. Journal of biomechanics, 40(5), 1058-1065.

Panjabi, M. M., 1992a. The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. Journal of spinal disorders, 5, 383-383.

Panjabi, M. M., 1992b. The stabilizing system of the spine. Part II. Neutral zone and instability hypothesis. Journal of spinal disorders, 5(4), 390.

Pleis, J. R., Ward, B., & Lucas, J., 2010. Summary health statistics for US adults: National Health Interview Survey, 2009. Vital and health statistics. Series 10, Data from the National Health Survey(249), 1.

Rogers, E. L., & Granata, K. P., 2006. Disturbed paraspinal reflex following prolonged flexion-relaxation and recovery. Spine, 31(7), 839.

Rosenbaum, D., & Hennig, E. M., 1995. The influence of stretching and warm up exercises on Achilles tendon reflex activity. Journal of Sports Sciences, 13(6), 481-490.

Safran, M. R., 1995. Elbow injuries in athletes: a review. Clinical orthopaedics and related research(310), 257.

Sbriccoli, P., Solomonow, M., Zhou, B. H., & Lu, Y., 2007. Work to rest durations ratios exceeding unity are a risk factor for low back disorder; a feline model. Journal of Electromyography and Kinesiology, 17(2), 142-152.

Sekine, M., Yamashita, T., Takebayashi, T., Sakamoto, N., Minaki, Y., & Ishii, S., 2001. Mechanosensitive afferent units in the lumbar posterior longitudinal ligament. Spine, 26(14), 1516.

Solomonow, M., 2009. Ligaments: a source of musculoskeletal disorders. Journal of bodywork and movement therapies, 13(2), 136-154.

Solomonow, M., 2012. Neuromuscular manifestations of viscoelastic tissue degradation following high and low risk repetitive lumbar flexion. Journal of Electromyography and Kinesiology, 22, 155-175.

Solomonow, M., Baratta, R., Zhou, B. H., Burger, E., Zieske, A., & Gedalia, A., 2003a. Muscular dysfunction elicited by creep of lumbar viscoelastic tissue. Journal of Electromyography and Kinesiology, 13(4), 381-396.

Solomonow, M., Baratta, R. V., Banks, A., Freudenberger, C., & Zhou, B. H., 2003b. Flexion-relaxation response to static lumbar flexion in males and females. Clinical Biomechanics, 18(4), 273-279.

Solomonow, M., Zhou, B. H., Baratta, R., Lu, Y., & Harris, M., 1999. Biomechanics of increased exposure to lumbar injury caused by cyclic loading: Part 1. Loss of reflexive muscular stabilization. Spine, 24(23), 2426.

Soslowsky, L., Thomopoulos, S., Tun, S., Flanagan, C., Keefer, C., Mostow, J., et al., 2000. Overuse activity injures the supraspinatus tendon in an animal model: A histologic and biomechanical study. Journal of Shoulder and Elbow Surgery, 9(2), 79-84.

Wai, E., Roffey, D., Bishop, P., Kwon, B., & Dagenais, S., 2010. Causal assessment of occupational lifting and low back pain: results of a systematic review. The Spine Journal, 10(6), 554-566.

Weir, D. E., Tingley, J., & Elder, G. C. B., 2005. Acute passive stretching alters the mechanical properties of human plantar flexors and the optimal angle for maximal voluntary contraction. European Journal of Applied Physiology, 93(5), 614-623.

Wilson, S. E., & Granata, K. P., 2003. Reposition sense of lumbar curvature with flexed and asymmetric lifting postures. Spine, 28(5), 513.

Zhang, L. Q., Huang, H., Sliwa, J. A., & Rymer, W. Z., 1999. System identification of tendon reflex dynamics. Rehabilitation Engineering, IEEE Transactions on, 7(2), 193-203.

6. Aim 4: Accumulation of Neuromuscular Disturbances from Repetitive Lifting

Toosizadeh N, Bazrgari B, Hendershot B, Muslim K, Nussbaum MA, Madigan ML: [ND] Disturbance and recovery of trunk mechanical and neuromuscular behaviors following repetitive lifting: influences of flexion angle and lift rate on creep-induced effect. Ergonomics, In Preparation.

Abstract
Repetitive lifting is associated with an increased risk of occupational low back disorders, yet potential adverse effects of such exposure on trunk mechanical and neuromuscular behaviors are not well described. Here, 12 participants, gender balanced, completed 40 minutes of repetitive lifting in all combinations of three flexion angles (33, 66, and 100% of each participant's full flexion angle) and two lift rates (2 and 4 lifts/min). Trunk behaviors were obtained pre- and post-exposure and during recovery using sudden perturbations. Intrinsic trunk stiffness and reflexive responses were compromised following lifting exposures, with larger decreases in stiffness and reflexive force caused by larger flexion angles. Larger flexion angles also delayed reflexive responses. Consistent effects of lift rate were not found. Except for reflex delay, no measures returned to pre-exposure values after 20 minutes of recovery. Simultaneous changes in both trunk stiffness and neuromuscular behaviors may impose an increased risk of trunk instability and low back injury. Practitioner Summary: An elevated risk of low back disorders is attributed to repetitive lifting. Here, the effects of flexion angle and lift rate on trunk mechanical and neuromuscular behaviors were investigated. Increasing flexion angle had adverse effects on these outcomes, though lift rate had smaller effects, and recovery time was >20 minutes.

Introduction
Low back disorders (LBDs) are among the most prevalent occupational injuries, involving annual costs in excess of $10 billion dollars for treatment in the United States alone (Martin et al. 2009). While diverse risk factors for LBDs have been identified, performing repetitive lifting tasks involving trunk flexion is associated with a particularly high risk (Kuiper et al. 1999, Hoogendoorn et al. 2000). Epidemiological studies have also provided evidence of increased LBD risk due to repetitive lifting in several occupational sectors, such as automobile industries and parcel delivery (Punnett et al. 1991, Prado-Leon et al. 2005). Other studies have suggested that lifting conditions, specifically trunk flexion angle and lift rate, can influence the risk of LBDs (Stobbe et al. 1988, Dolan et al. 1994, Lin et al. 2002). However, the underlying mechanism(s) linking these lifting conditions and LBD development are still unclear.

Experimental studies have demonstrated changes in mechanical and neuromuscular properties of the trunk due to repetitive flexion (which is required typically when performing lifting tasks). The most commonly reported mechanical consequence of repetitive trunk flexion is a reduction in passive stiffness of the trunk (Parkinson et al. 2004, Olson et al. 2009, Shin and D'Souza 2010), and which is likely subsequent to changes in viscoelastic behaviors of spinal motion segments and passive muscle components. Cyclic flexion leads to creep and load-relaxation of human lumbar motion segments (Little and Khalsa, 2005), and cyclic elongation of muscle-tendon units beyond resting length can lead to a ~15% reduction in peak tensile forces after 10 cycles (Taylor et al. 1990, Magnusson et al. 2000). In addition to these changes in the mechanical properties of the trunk, repetitive flexion can also cause neuromuscular alterations, such as muscle spasms and compromised reflex responses of paraspinal muscles (Claude et al. 2003). Such neuromuscular alterations could subsequently impair the stability of the trunk

(Panjabi 1992, Panjabi 2003). Moreover, recovery of trunk mechanical and neuromuscular behaviors may differ depending on the specific pattern of flexion exposures. For example, in our previous work we found that mechanical properties recovered faster than neuromuscular behaviors following prolonged trunk flexion at a constant angle (load-relaxation: Hendershot et al. 2011); however, opposing results (i.e., faster recovery of neuromuscular behaviors) were found after prolonged passive trunk flexion (creep: Bazrgari et al. 2011a).

While this existing evidence suggests mechanical and neuromuscular alterations in trunk behaviors following cyclic loading, it remains to determine whether/how such changes occur in the human trunk while performing an actual lifting task and the potential modifying effects of specific task demands. Accordingly, the goal of this study was to evaluate the effects of repetitive lifting on mechanical and neuromuscular behaviors of the trunk. We have reported previously that the stiffness and reflex response of the trunk are more substantially affected following exposure to larger trunk flexion angles (Hendershot et al. 2011). In addition, changes in mechanical and neuromuscular behaviors in response to repetitive flexion exposures (in a feline model) are frequency dependent (Lu et al. 2008). As such, we hypothesized that: 1) the magnitude of alterations in trunk mechanical and neuromuscular behaviors during repetitive lifting increases with trunk flexion angle and lift rate, and 2) patterns of recovery are different between mechanical and neuromuscular behaviors.

Methods

Participants

Twelve healthy young adults, with no self-reported history of low-back pain or current medical conditions, completed the study after providing informed consent. All experimental procedures were approved by the Virginia Tech Institutional Review Board. Participants included six males, with respective mean (SD) age, stature, and body mass of 22 (3) yr, 182.1 (3.8) cm, and 75.9. (6.3) kg; corresponding values for the six females were 24 (3) yr, 165.2 (4.4) cm, and 59.1 (5.9) kg. A relatively young group of participants (from 19-28 yr) was included to avoid potential influences related to age.

Experimental design and procedures

A repeated-measures design was used, in which several measures of trunk mechanical and neuromuscular behaviors were obtained prior to, during, and following repetitive dynamic lifting. There were six different lifting conditions, involving all combinations of three flexion angles (33, 66, and 100% of each participant's full flexion angle) and two lift rates (2 and 4 lifts/min). These lifting conditions were intended to cover, respectively, a wide range of potential exposures involving passive tissue strain and both low- and high-risk lifting rates (Marras et al., 1993). Sessions were conducted at a similar time on separate days, with at least 72 hours between consecutive sessions. The presentation order of conditions was counterbalanced using two 6 x 6 Latin Squares (one for each gender) to reduce potential order-related confounding effects.

Electromyography (EMG) of the erector spinae (at the L1 and L3 levels), rectus abdominus, and external oblique muscles was collected bilaterally using bipolar Ag/AgCl surface electrodes, with electrode placements as previously reported (McGill 1991, Larivière et al. 2009, Bazrgari et al. 2011a, Hendershot et al. 2011). Raw EMG signals were pre-amplified (x100) near the collection site, bandpass filtered (10-500Hz), amplified (x100), and converted to root mean square (RMS; time constant = 110 msec) in hardware (Measurement Systems Inc., Ann Arbor, MI, USA). To measure trunk flexion angle, a triaxial 6 DOF inertial measurement unit (IMU: Xsens Technologies XM-B-XB3, Enschede, Netherlands) was placed over the T12 vertebral process using medical-grade, double-sided tape. EMG signals were sampled at 1000Hz and the IMU at 100 Hz.

After instrumentation, and at the beginning of the first session, three trials were performed to record the lumbar flexion angle at full trunk flexion. Participants stood in a rigid metal frame and straps were used to restrain the pelvis and lower limbs (Figure 1). Subsequently, they slowly flexed forward from upright standing to full flexion (passive hanging position), with minimal muscle activity and their head facing down and their arms relaxed and hanging vertically. Participants remained in the flexed posture for ~5 seconds, during which lumbar flexion angle was measured. Since the pelvis was restrained the angle measured from the IMU at T12 represented the lumbar flexion angle. The mean lumbar flexion angle across the three trials was obtained as the full lumbar flexion angle (FLFA) for each participant. Next, three trials of maximum voluntary contraction (MVC) were performed in neutral standing posture to assess trunk extension strength. During these, a rigid rod and chest harness assembly (Figure 1) was used, and participants pulled back maximally on the rod for 5 seconds. Muscle activity (EMG) was collected and processed as described earlier, and force was measured (1000 Hz) using a load cell (Interface SM2000, Scottsdale, AZ, USA) on the harness-rod assembly. The maximum force and peak EMG values for each muscle were identified across the three trials, and were used subsequently for normalization (see below).

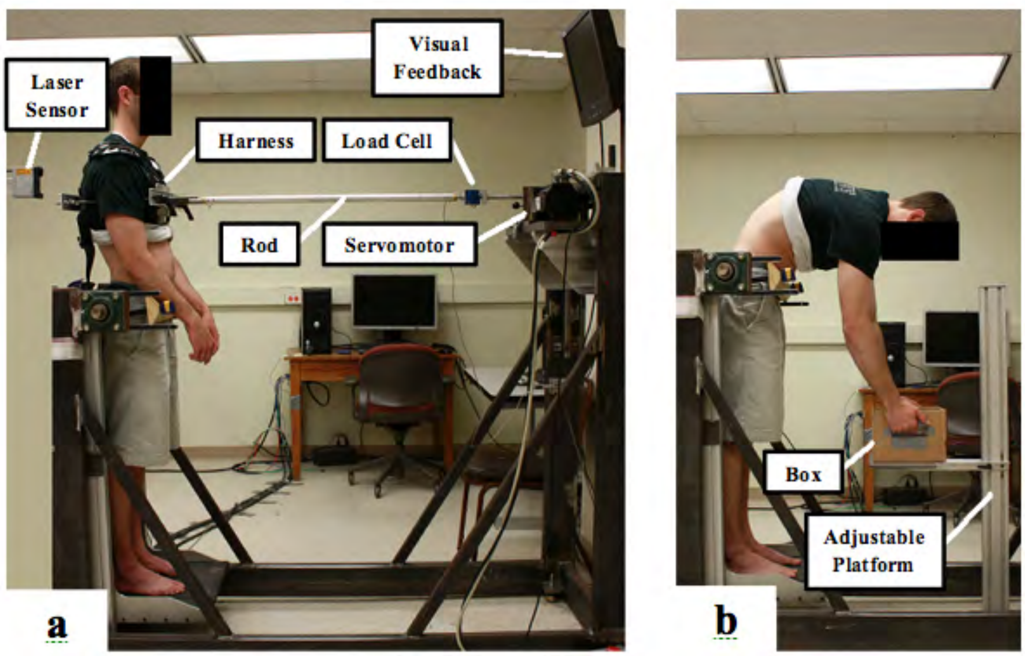

Figure 1. Experimental setup, demonstrating a participant during: a) sudden perturbation procedure (the same setup was used to perform MVC and fatigue tests), and b) the start of a lifting effort.

Repetitive lifting was performed for 40 minutes at a specific rate (i.e., either 2 or 4 lifts/min) that was timed by a digital metronome. The 40-minute duration was based on the results of Parkinson et al. (2004), who observed significant spinal creep after 30 minutes of repetitive flexion. For each lift, participants started from an upright standing posture, bent forward to grasp a 29N box located on a platform in front of their legs, lifted to an upright posture, placed the box back on the platform, and then returned to an upright posture between lifts. Box weight here correspond to values handled in low-risk occupational manual material handling tasks (Marras et al. 1993), represented roughly the 15th percentile of typical handled loads (Ciriello and Snook 1999), and was kept relatively low to minimize the effect of extensor muscle fatigue. For each participant, platform height was adjusted so that the peak trunk flexion angle necessary to grasp the box handles was equivalent to 33%, 66%, or 100% of the participant's FLFA. Following the period of repetitive lifting, participants maintained an upright standing posture, while constrained in the frame, for 20 minutes to assess immediate post-exposure effects and recovery.

Trunk behaviors were obtained using a sudden-perturbation paradigm, and following identical data collection and analysis procedures reported in our earlier studies (Bazrgari et al. 2011a, Hendershot et al. 2011, Miller et al. 2012) (Muslim et al., 2012). Briefly, these measures were collected while participants were in an upright posture, during which a 45 s sequence of 12 small (±5 mm), rapid (< 40 ms) anterior–posterior perturbations were imposed to the trunk via a servomotor, rigid rod and chest harness (Figure 1). Postural displacements were measured with a laser displacement sensor (Keyence LK-G 150, Osaka, Japan) and the motor encoder, while applied forces were measured using the in-line load cell. Prior to and during the perturbation sequences, participants maintained a constant submaximal extensor effort. The target effort was set to 10% of maximum voluntary RMS EMG in the bilateral L3 erector spinae. Measurements of mechanical and neuromuscular behaviors were recorded before, during (at 5, 10, 20, 30, and 40 min), and after (at 2.5, 5, 10, and 20 min) the repetitive lifting task (Figure 2). Short delays (~1 min) within the total lifting sequence were required to attach and remove the measurement equipment, and to complete the perturbation sequence. Isometric reference contractions (to assess fatigue) were performed before the lifting task, after the task (after the final perturbation measurement at 40 minutes), and after recovery (after 20 min of standing). Reference contractions involved maintaining a sub-maximal extensor force of 50% MVC force for 30 seconds, maintained using real-time visual feedback of the force (Roy et al. 1989, Dolan and Adams 1998).

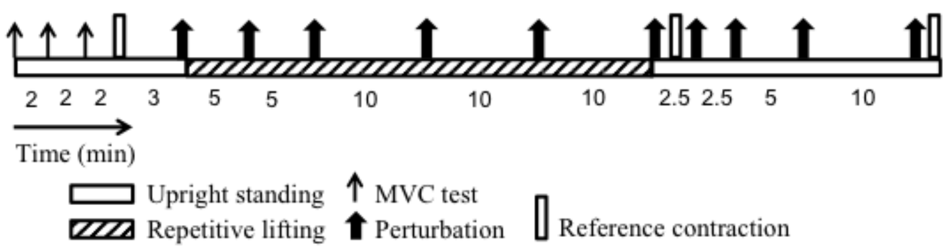

Figure 2. An overview of the experimental procedures, including pre-exposure measurements (~10 min), exposure to repetitive lifting (~40 min), and a post-exposure recovery period (~20 min). Time intervals between selected measurement/tests are indicated.

Outcome measures
Similar to our previous studies (Bazrgari et al. 2011a, Hendershot et al. 2011, Miller et al. 2012) (Muslim et al., 2012), several outcome measures were obtained to characterize trunk mechanical and neuromuscular behavior: 1) apparent mass and intrinsic trunk stiffness; 2) reflex delay; 3) maximum reflex force; and 4) timing of maximum reflex force. In addition, changes in erector spinae EMG were measured to assess muscle fatigue. The latency of reflexive muscle response (i.e., reflex delay) was determined as the time delay between an anteriorly-directed perturbation and the onset of erector spinae muscle reflex response (Zhang et al. 1999, Granata et al. 2005). Intrinsic trunk stiffness (due to both passive tissue and baseline muscle activation) was quantified from the trunk dynamic response to the applied perturbations in a predefined time window, which started from the onset of an anterior

perturbation and ended at the reflex onset of the erector spinae musculature. Parameters of a model representing the trunk (apparent mass and intrinsic trunk stiffness) were estimated using a least-squares curve fit in MATLABTM (MathWorks, Natick, MA, USA); this was done separately for each anteriorly-directly perturbation by relating measured trunk kinematics to trunk kinetics (both measured in the horizontal direction at T8).

To characterize trunk neuromuscular behaviors, reflex forces were first estimated by subtracting the model-estimated intrinsic force contribution from the total measured trunk response (i.e., trunk reaction force measured by the inline load cell). Magnitude and timing (with respect to perturbation onset) of the maximum reflex force were quantified to represent the overall trunk reflexive behavior (Bazrgari et al. 2011b). For each anteriorly-directed perturbation, the analysis was limited to a time window of 150 ms following reflex onset to avoid voluntary muscle responses (Bazrgari et al. 2011a, Hendershot et al. 2011) (Muslim et al., 2012). From the three reference contractions, mean values of EMG RMS and median power frequency (MF) of the extensor muscles (erector spinae muscle at the L1 and L3 levels) were derived after normalization, and following similar procedures described earlier (Dolan and Adams 1998).

Analysis

Pre-exposure differences between genders in apparent mass, intrinsic trunk stiffness, reflex delay, and the magnitude and timing of the maximum reflex force were evaluated using separate single-factor analyses of variance (ANOVA). Paired t tests were used to assess overall (across all conditions) changes in these measures immediately following the exposure period. Subsequently, all post-exposure measures were normalized to pre-exposure values [(post-pre)/pre], and the acute effects of flexion angle, lift rate, and gender were assessed using mixed-factor analyses of variance (ANOVAs). A similar approach, using ANOVAs, was used to assess the effect of flexion angle and lift rate at different time periods during the lifting task.

Additional ANOVAs were used to assess changes in several measures during the exposure (or recovery) periods: 1) changes in the total trunk extensor muscle activation (maximum EMG RMS) during the last three vs. first three lifts; 2) changes in the total submaximal extensor efforts (mean EMG RMS) and total flexor muscle antagonistic co-contraction (mean EMG RMS) generated in the last vs. first perturbation sequences; 3) fatigue, as determined by changes in mean EMG RMS and mean MF of the trunk extensor muscles during the reference contractions. To achieve this, “time” (i.e., pre-exposure, and post-exposure, and recovery for reference contractions) was defined as an additional independent variable. As relevant, post hoc pairwise comparisons were performed using Tukey’s Honestly Significant Difference (HSD) tests. All analyses were done using JMPTM (Version 8, SAS Institute Inc., Cary, NC), and statistical significance was concluded when $p < 0.05$. Summary results are presented as means (SDs). Reflex delay measures from one condition of one participant (66% FLFA and 4 lifts/min) were excluded due to an inability to capture EMG-based reflex responses consistently.

Results

3.1. Pre-exposure

Pre-exposure apparent mass and intrinsic trunk stiffness were significantly larger among males than females (Table 1). All measures of trunk reflexive behavior (reflex delay and the magnitude and timing of maximum reflex force) were comparable between genders.

Table 1. Pre-exposure measures of trunk behaviors. Means (SDs) are provided, and asterisks indicate significant differences between genders.

Measure	Male	Female	ANOVA	*p* value
Apparent mass (kg)	20.3 (3.4)	16.7 (3.0)	$F_{(1,10)} = 5.6$	0.040 *
Intrinsic trunk stiffness (N/m)	8968 (1039)	6139 (852)	$F_{(1,10)} = 42.2$	< 0.0001 *
Reflex delay (ms)	62.4 (4.5)	60.7 (5.5)	$F_{(1,9)} = 0.5$	0.48
Max. reflex force (N)	194 (36)	193 (46)	$F_{(1,10)} = 0.004$	0.95
Timing of Max. reflex force (ms)	160.4 (8.2)	157.1 (8.3)	$F_{(1,10)} = 0.8$	0.38

Post-exposure
Intrinsic trunk stiffness significantly decreased by 527 (541) N/m across all exposure conditions (Table 2), and the effect of exposure was significantly larger with increased flexion angle (Figure 3). There was a significant increase in reflex delay post-exposure. While the mean difference was small (1.2 (3.9) msec), it also was significantly larger with increased flexion angle (Figure 3). Both the magnitude and timing of maximum reflex force significantly changed post-exposure, respectively decreasing and increasing; pre- vs. post-exposure values of maximum reflex force were 193 (41) vs. 167 (44) N, and 159 (8) vs. 161 (12) msec for the timing of maximum reflex force. These changes were also significantly affected by flexion angle, with larger changes following exposure to increased angle (Figure 3). However, flexion angle had no interactive effect on any of the outcome measures (p > 0.11). There were no main effects of lift rate, though the effect on changes in maximum reflex force approached significance (p = 0.051), with respective decreases of 20 (28) and 33 (28) N in the 2 and 4 lifts/min conditions. There were also no main effects of gender on any of the outcome measures. No interactive effects involving lift rate or gender were significant (p > 0.11), except for a gender × lift rate effect on maximum reflex force (p = 0.030). Regarding the latter, females had a more substantial reduction in maximum reflex force in response to the higher lift rate, whereas the reduction among males was comparable between the two lift rates.

Table 2. Effects of repetitive lifting on trunk behaviors. The '"Overall difference" column indicates whether there were significant post- vs. pre-exposure changes across all conditions. Main effects of experimental conditions are also indicated, with significant effects indicated by asterisks.

Measure	Overall difference	Main Effect		
		Flexion angle	Lift rate	Gender
Intrinsic trunk stiffness	$t_{(71)} = -8.3, p < 0.0001$ *	$F_{(2,45)} = 5.9, p = 0.0051$ *	$F_{(1,45)} = 0.1, p = 0.75$	$F_{(1,10)} = 0.5, p = 0.49$
Reflex delay	$t_{(70)} = 2.7, p = 0.010$ *	$F_{(2,44)} = 4.0, p = 0.025$ *	$F_{(1,44)} = 2.8, p = 0.10$	$F_{(1,10)} = 0.8, p = 0.38$
Max. reflex force	$t_{(71)} = -6.6, p < 0.0001$ *	$F_{(2,45)} = 3.6, p = 0.034$ *	$F_{(1,45)} = 4.0, p = 0.051$	$F_{(1,10)} = 2.9, p = 0.12$
Timing of Max. reflex force	$t_{(71)} = 2.5, p = 0.016$ *	$F_{(2,45)} = 11.3, p = 0.0002$ *	$F_{(1,45)} = 0.1, p = 0.74$	$F_{(1,10)} = 1.9, p = 0.20$

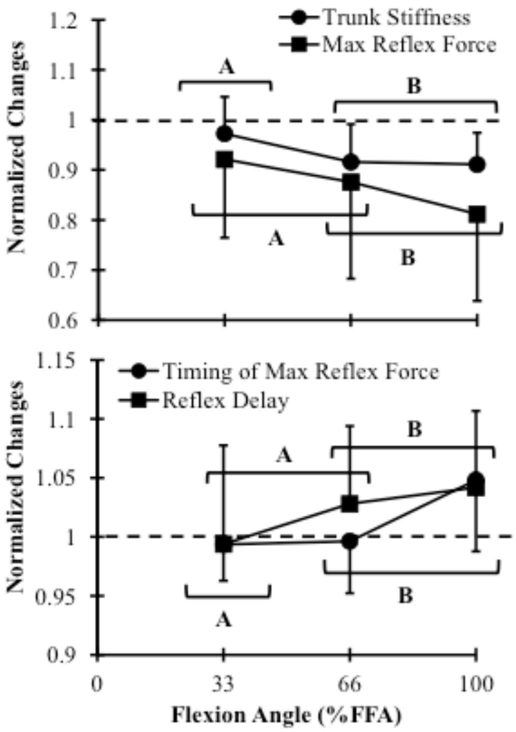

Figure 3. Effects of flexion angle on intrinsic trunk stiffness, the magnitude and timing of maximum reflex force, and reflex delay. Normalized changes are indicated (e.g., a value of 1.05 indicates a 5% increase from pre-exposure levels). Error bars indicate standard deviations, and post-hoc groupings are indicated by brackets and letters. (FLFA = full flexion angle).

Total trunk extensor muscle activity (EMG RMS) increased ~5% during lifting at the end of the exposure period (i.e., last vs. first three lifts), however the main effect of time was not significant ($p = 0.73$). EMG RMS values were comparable between the last and first perturbation trials during the lifting exposures ($p = 0.16$ for total extensor muscle EMG RMS, and $p = 0.99$ for total flexor muscle EMG RMS). There were no significant changes in EMG RMS or MF values during the reference contractions performed pre- and post-exposure or following recovery ($p > 0.54$). Moreover, there was no interactive effect of time with flexion angle, lift rate, and gender for any of these outcome measures ($p > 0.19$).

Recovery

Post-exposure reductions in intrinsic trunk stiffness recovered by ~43% after 20 minutes of upright standing (Figure 4), whereas changes in reflex delay were recovered by ~70% after the same recovery period. In contrast, recovery of maximum reflex force was slower, with an overall ~14% increase compared to post-exposure values. No recovery was evident for the timing of maximum reflex force, which instead continued to increase during the recovery period. Comparisons between initial (pre-exposure) and final (post-recovery) values demonstrated that only reflex delay was fully recovered ($t(68) = 1.01$, $p = 0.32$), whereas the other outcome measures were still significantly different from pre-exposure values ($p < 0.012$). Qualitatively, no consistent differences in recovery behaviors were evident between flexion angle exposures for any of the outcome measures (Figure 4).

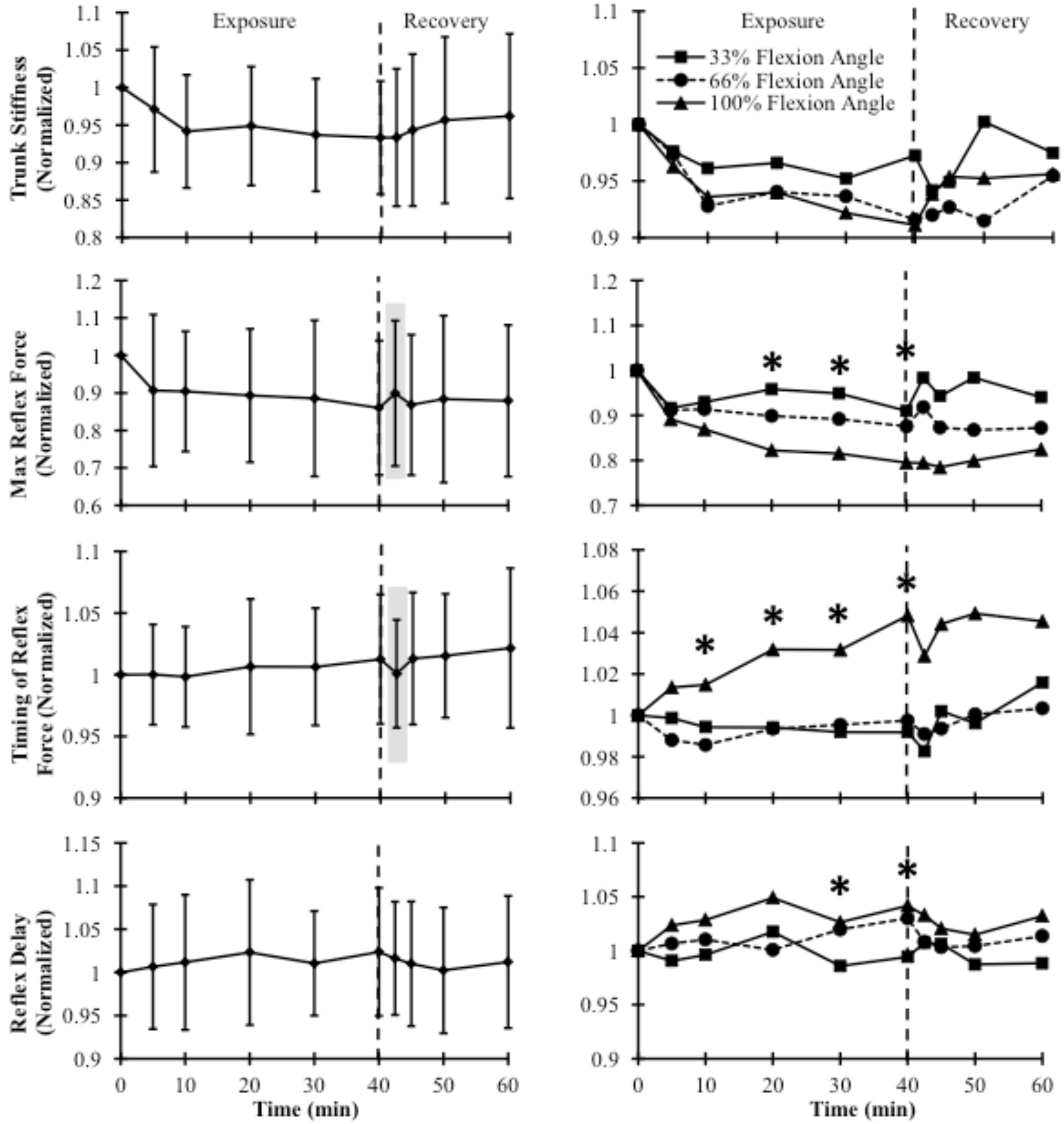

Figure 4. Normalized changes in trunk behaviors (intrinsic trunk stiffness, magnitude and timing of maximum reflex force, and reflex delay) during the exposure and recovery periods (left), and the effect of flexion angle on the same parameters (right). Error bars indicate standard deviations, and asterisks indicate significant effects of flexion angle at a given time. An apparent hyperexcitability during recovery is highlighted with grey bars.

Discussion

Intrinsic trunk behavior

Intrinsic trunk stiffness, which here represents the overall contributions of passive trunk tissues and baseline muscle activation around the neutral standing posture, decreased by ~7% across all lifting conditions after 40 minutes of repetitive lifting. Muscular fatigue is a potential confounding influence, yet there was no evidence of fatigue following the lifting task (i.e. based on EMG RMS and MF). Further, baseline muscle activation, another potential confounding effect, was not significantly changed between initial and final perturbations. Baseline muscle activation during perturbation, although it was not significant, actually increased by ~4% following lifting across all exposure conditions. As such, the observed decrease in stiffness here likely resulted from reductions in passive contributions to intrinsic trunk stiffness, and may slightly underestimate actual decreases in stiffness. Previous in vivo studies have similarly reported reductions in passive stiffness of the human trunk after repetitive flexion/lifting exposures (Olson et al. 2004, Parkinson et al. 2004, Olson et al. 2009). Noticeable creep deformation was also observed in studies of repetitive flexion exposure using feline spines (Hoops et al. 2007, Solomonow 2011), and which can be interpreted as a reduction in rotational stiffness. Although repetitive lifting is different from repetitive flexion, similar viscoelastic deformation and consequently reduction in passive trunk stiffness are expected due to stretching/deformation of soft tissues.

Changes in intrinsic trunk stiffness caused by repetitive lifting were more substantial with larger trunk flexion angles (Figures 3 and 4). Our earlier results for prolonged trunk flexion exposures also demonstrated a larger decrease in intrinsic trunk stiffness with increasing flexion angle (Hendershot et al. 2011); however, there is a subtle but notable difference in these effects of flexion angle. In the current study, reductions in intrinsic trunk stiffness were almost equal in response to exposures involving 66 and 100% of full flexion (Figure 3), suggesting an asymptote at some point in the middle of the trunk range of motion. In the earlier study of prolonged trunk flexion, the results suggested increasing effects up to the full flexion exposure. One contribution to this difference is likely the way in which the maximum (100%) flexion exposure level was achieved. Here, the maximum trunk flexion angle was used, whereas in previous study the flexion-relaxation angle was used. As such, maximum trunk flexion angle was larger here, leading to a larger range of flexion angle exposures.

Qualitatively, alterations in intrinsic trunk stiffness during both the exposure and rest periods showed exponential behaviors (Figure 4), similar to those reported earlier (McGill and Brown 1992, Youssef et al. 2008, Solomonow 2011). About 87% of the decrease in intrinsic trunk stiffness occurred in the first 10 minutes of exposure, and ~81% of the total recovery occurred within the first 10 minutes of the standing recovery period. Further, the rate of recovery of intrinsic trunk stiffness appeared to be slower here compared to the rate of changes during exposure, which is also consistent with previous experiments (Hoops et al. 2007, Sbriccoli et al. 2007, Solomonow 2011).

Based on earlier results, spine rotational stiffness and stability are closely related (Arjmand and Shirazi-Adl 2006, Bazrgari and Shirazi-Adl 2007, Graham and Brown 2012), and the reductions in passive stiffness observed here could compromise trunk stability and increase the risk of LBDs (Hoogendoorn et al. 2000). To stabilize the trunk, an increase in paraspinal muscle activation may be required to compensate for reductions in passive stiffness (Marras and Granata 1997, Olson et al. 2009). We observed that the total trunk extensor muscle activation (EMG RMS) increased during lifts performed at the end vs. beginning of the exposure periods. Although the increases were small (~5% across all exposure conditions), and not statistically significant, this effect may still be of relevance given that the moment arms of paraspinal muscles are also relatively small. As such, even small increases in muscle forces (which is considered to be the case given a lack of evidence for muscle fatigue) could result in important increases in spine loads (e.g., compressive forces).

Trunk neuromuscular behavior

Repetitive lifting reduced and delayed maximum reflex forces. Three main factors have been hypothesized to influence muscle reflexive behavior: laxity in viscoelastic tissues, fatigue, and habituation (Solomonow et al. 1999, Granata et al. 2005, Jackson et al. 2009, Bazrgari et al. 2011a, Hendershot et al. 2011). Here, EMG-based measures provided no evidence for muscle fatigue, and the effect of habituation was likely minimized using pseudorandom timing of the perturbations. As such, the observed changes in trunk muscle reflexive behaviors likely arose from strains in ligaments and other soft tissues. Reductions in reflex force due to strains in ligaments were observed in earlier work, which showed that reflexive activities of the feline multifidus decreased during/following repetitive loading exposures (Solomonow et al. 1999). Comparable changes in the magnitude and timing of reflex responses have also been observed in human skeletal muscles surrounding other joints following passive stretching (Rosenbaum and Hennig 1995, Avela et al. 1999).

Similar to intrinsic trunk stiffness, trunk reflexive behaviors were significantly influenced by trunk flexion angle (Figures 3 and 4). While trunk flexion angle during lifting is an important factor in work design, no study has, to our knowledge, investigated the effect of this factor on trunk behaviors. Overall, the current results indicate an adverse effect of increased trunk flexion angle during repetitive lifting on reflexive and mechanical behaviors of the trunk. Regarding recovery behaviors reflex delay and maximum reflex force tended to recover during 20 minutes of standing; in contrast, the timing of reflex force presented an increasing trend even during the recovery period (Figure 4). There was an apparent muscular hyperexcitability in the first measures during the recovery period (i.e., Figure 4 at 42.5 min), which are noticeable as respective increases and reductions in the magnitude and timing of maximum reflex force, immediately after the exposure period. Similar behavior was also observed using feline spines, and was suggested as a protective mechanism to compensate for reductions in intrinsic trunk stiffness immediately after flexion exposures (Claude et al. 2003, Solomonow 2011).

Although not statistically significant, reductions in the maximum reflex force tended to be larger with a higher lift rate. Prior evidence suggests increased creep and reductions in trunk reflexive behaviors with an increasing rate of repetitive flexion (Lu et al. 2008). However, the lifting task here involved active muscle activity, as opposed to repetitive passive trunk flexion as in Lu et al. (2008). Moreover, there is a higher likelihood of extensor muscle fatigue with increasing lift rate (Kim and Chung 1995). Therefore, high lift rates were avoided here to prevent extensor muscle fatigue and potential confounding effects on the outcome measures.

Repetitive vs. prolonged strain of trunk soft tissues appears to result in divergent influences on trunk reflex force. In contrast to repetitive lifting (current study) and repeated static flexion (Muslim et al., 2012), prolonged flexion exposures leads to an increase in post-exposure maximum reflex force (Bazrgari et al. 2011a, Hendershot et al. 2011). Although an adverse effect of trunk flexion on reflexive behavior is apparent, the underlying mechanisms that can lead to either larger or smaller reflex forces are still unknown. Further, the timing of maximum reflex force and reflex delay increased here following repetitive lifting. In our current work, the timing of maximum reflex force and reflex delay were estimated by different methods, respectively using measured force and EMG, and consistency between these results provides some level of consensual validity. Generally, reflex responses of the trunk muscles play an important role in controlling the stability of the spine, and with less energy expenditure compared to voluntary co-contraction of trunk muscles (Franklin and Granata 2007, Moorhouse and Granata 2007). Therefore, any alteration in reflexive behaviors, as found in the current study, could compromise the efficiency of the neuromuscular system in stabilizing the spine. Of note, higher reflex forces following prolonged flexion exposures in our earlier work (Bazrgari et al. 2011a, Hendershot et al. 2011) were suggested as a compensatory mechanism for intrinsic trunk stiffness reduction. Yet as found in the current study and our earlier investigation of repetitive static flexion (Muslim et al., 2012), concurrently reduced stiffness and reflex force could impose a higher risk of LBDs.

Implications, Limitations, and Conclusions

Generally, and in support of our first hypothesis, the present results indicate adverse effects of larger trunk flexion angles during lifting on both mechanical and neuromuscular properties of the trunk. Except for maximum reflex force, which decreased by ~10% across all conditions following lifting task, all outcome measures showed no substantial changes for lifting from heights associated with 33% of maximum trunk flexion angle. This suggests that some potential adverse effects of repetitive lifting could be avoided by controlling lifting height (i.e., to result in trunk flexion less than ~one-third of flexion range-of-motion). Limited evidence was found to suggest that a reduction in lift rate would have similar beneficial effects.

Overall, recovery (to pre-exposure values) was faster for mechanical properties compared to recovery of the magnitude and timing of maximum reflex force (i.e., supporting the second hypothesis). Further, although the period of recovery here was only 20 minutes, trends in the outcome measures (except for reflex delay, which had a more rapid recovery) suggest that full recovery requires a longer recovery time vs. the exposure time. As such, work-rest cycles with duty cycles ≥ 50% may not be adequate to prevent the accumulation of mechanical and neuromuscular disturbances during occupational tasks requiring repetitive lifting. However, additional studies are required to determine more specifically what rest periods or duty cycles would be sufficient. For example, the current work involved exposures that were limited to 60 minutes to minimize potential confounding effects of prolonged standing posture on trunk behaviors (e.g., axial creep and muscle fatigue). Future studies should consider longer work/rest periods, a larger range of lift rates, and more realistic working conditions.

One potential limitation in our data analyses is related to the method used for estimating intrinsic trunk stiffness. As we have discussed previously (Bazrgari et al. 2011a, Hendershot et al. 2011) (Muslim et al., 2012), measured reductions in intrinsic stiffness might be conservative (underestimated), since measurements were performed in a posture (neutral standing) that is associated with the lowest intrinsic trunk stiffness (Parkinson et al. 2004, Shirazi-Adl 2006). Further, trunk damping has recently been suggested as a means for better understanding spinal stability and neuromuscular control (Cholewicki et al. 2005); however, we forced trunk damping to zero to prevent our model predictions of the dynamic responses of the trunk to be dominated by damping. This simply transferred the predominant response from damping to stiffness, to facilitate comparisons with existing literature. However, some caution is warranted when interpreting the relative contribution of elastic and viscous components here and in earlier work, since the models used likely represent oversimplifications of the spine, and predicted properties may not be the best physical representation of the system.

In summary, the present work provides experimental evidence of mechanical and neuromuscular alterations in the human trunk caused by repetitive lifting. Our earlier results indicated an increase in reflex force following prolonged trunk flexion, and this increase was suggested to compensate for decreases in passive trunk stiffness (Bazrgari et al. 2011a, Hendershot et al. 2011). Alterations in passive trunk stiffness and reflex responses here indicate a deterioration in both behaviors following a repetitive lifting task. Consequently, simultaneous changes in both passive trunk stiffness and neuromuscular behaviors can be interpreted as inducing a higher risk of LBD when performing demanding tasks (such as lifting heavy weights) following repetitive vs. prolonged trunk flexion exposures. As such, and in agreement with previous suggestions (McGill 2007), it may be important in job design to avoid demanding tasks after repetitive lifting unless sufficient rest/recovery is provided.

Acknowledgments

This work was supported by an award (R01OH008504) from the Centers for Disease Control and Prevention (CDC). The contents are solely the responsibility of the authors and do not necessarily represent the official views of the CDC. The authors thank Mr. Waldron and Mr. Vest for their assembly and maintenance of the testing apparatus. The authors dedicate this work to Dr. Kevin P. Granata, whose original ideas formed the foundation for the current investigation.

References

Arjmand, N. & Shirazi-Adl, A., 2006. Model and in vivo studies on human trunk load partitioning and stability in isometric forward flexions. Journal of Biomechanics, 39 (3), 510-521.

Avela, J., Kyröläinen, H. & Komi, P.V., 1999. Altered reflex sensitivity after repeated and prolonged passive muscle stretching. Journal of Applied Physiology, 86 (4), 1283-1291.

Bazrgari, B., Hendershot, B., Muslim, K., Toosizadeh, N., Nussbaum, M.A. & Madigan, M.L., 2011a. Disturbance and recovery of trunk mechanical and neuromuscular behaviours following prolonged trunk flexion: Influences of duration and external load on creep-induced effects. Ergonomics, 54 (11), 1043-1052.

Bazrgari, B., Nussbaum, M.A. & Madigan, M.L., 2011b. Estimation of trunk mechanical properties using system identification: Effects of experimental setup and modelling assumptions.

Bazrgari, B. & Shirazi-Adl, A., 2007. Spinal stability and role of passive stiffness in dynamic squat and stoop lifts. Computer Methods in Biomechanics and Biomedical Engineering, 10 (5), 351-360.

Cholewicki, J., Silfies, S.P., Shah, R.A., Greene, H.S., Reeves, N.P., Alvi, K. & Goldberg, B., 2005. Delayed trunk muscle reflex responses increase the risk of low back injuries. Spine, 30 (23), 2614.

Ciriello, V.M. & Snook, S.H., 1999. Survey of manual handling tasks. International Journal of Industrial Ergonomics, 23 (3), 149-156.

Claude, L.N., Solomonow, M., Zhou, B.H., Baratta, R.V. & Zhu, M.P., 2003. Neuromuscular dysfunction elicited by cyclic lumbar flexion. Muscle ad Nerve, 27 (3), 348-358.

Dolan, P. & Adams, M., 1998. Repetitive lifting tasks fatigue the back muscles and increase the bending moment acting on the lumbar spine. Journal of Biomechanics, 31 (8), 713-721.

Dolan, P., Earley, M. & Adams, M., 1994. Bending and compressive stresses acting on the lumbar spine during lifting activities. Journal of Biomechanics, 27 (10), 1237-1248.

Franklin, T.C. & Granata, K.P., 2007. Role of reflex gain and reflex delay in spinal stability—a dynamic simulation. Journal of Biomechanics, 40 (8), 1762-1767.

Graham, R.B. & Brown, S.H.M., 2012. A direct comparison of spine rotational stiffness and dynamic spine stability during repetitive lifting tasks. Journal of Biomechanics.

Granata, K.P., Rogers, E. & Moorhouse, K., 2005. Effects of static flexion-relaxation on paraspinal reflex behavior. Clinical Biomechanics, 20 (1), 16-24.

Hendershot, B., Bazrgari, B., Muslim, K., Toosizadeh, N., Nussbaum, M.A. & Madigan, M.L., 2011. Disturbance and recovery of trunk stiffness and reflexive muscle responses following prolonged trunk flexion: Influences of flexion angle and duration. Clinical Biomechanics, 26 (3), 250-256.

Hoogendoorn, W.E., Bongers, P.M., De Vet, H.C.W., Douwes, M., Koes, B.W., Miedema, M.C., Ariëns, G.a.M. & Bouter, L.M., 2000. Flexion and rotation of the trunk and lifting at work are risk factors for low back pain: Results of a prospective cohort study. Spine, 25 (23), 3087.

Hoops, H., Zhou, B.H., Lu, Y., Solomonow, M. & Patel, V., 2007. Short rest between cyclic flexion periods is a risk factor for a lumbar disorder. Clinical Biomechanics, 22 (7), 745-757.

Jackson, N.D., Gutierrez, G.M. & Kaminski, T., 2009. The effect of fatigue and habituation on the stretch reflex of the ankle musculature. Journal of Electromyography and Kinesiology, 19 (1), 75-84.
Kim, S. & Chung, M., 1995. Effects of posture, weight and frequency on trunk muscular activity and fatigue during repetitive lifting tasks. Ergonomics, 38 (5), 853-863.
Kuiper, J.I., Burdorf, A., Verbeek, J.H.a.M., Frings-Dresen, M.H.W., Van Der Beek, A.J. & Viikari-Juntura, E.R.A., 1999. Epidemiologic evidence on manual materials handling as a risk factor for back disorders: A systematic review. International Journal of Industrial Ergonomics, 24 (4), 389-404.
Larivière, C., Gravel, D., Gagnon, D. & Arsenault, A.B., 2009. Toward the development of predictive equations of back muscle capacity based on frequency-and temporal-domain electromyographic indices computed from intermittent static contractions. The Spine Journal, 9 (1), 87-95.
Lin, Y.H., Chen, C.S., Chen, W.J. & Cheng, C.K., 2002. Characteristics of manual lifting activities in the patients with low-back pain. International Journal of Industrial Ergonomics, 29 (2), 101-106.
Lu, D., Le, P., Davidson, B., Zhou, B.H., Lu, Y., Patel, V. & Solomonow, M., 2008. Frequency of cyclic lumbar loading is a risk factor for cumulative trauma disorder. Muscle ad Nerve, 38 (1), 867-874.
Magnusson, S.P., Aagaard, P. & Nielson, J.J., 2000. Passive energy return after repeated stretches of the hamstring muscle-tendon unit. Medicine and Science in Sports and Exercise, 32 (6), 1160.
Marras, W.S. & Granata, K.P., 1997. Changes in trunk dynamics and spine loading during repeated trunk exertions. Spine, 22 (21), 2564.
Marras, W.S., Lavender, S.A., Leurgans, S., Rajulu, S., Allread, W.G., Fathallah, F.A. & Ferguson, S.A., 1993. The role of dynamic three-dimensional trunk motion in occupationally-related low back disorders. Spine, 18 (5), 617-628.
Martin, B.I., Turner, J.A., Mirza, S.K., Lee, M.J., Comstock, B.A. & Deyo, R.A., 2009. Trends in health care expenditures, utilization, and health status among us adults with spine problems, 1997–2006. Spine, 34 (19), 2077.
Mcgill, S., 2007. Low back disorders: Evidence-based prevention and rehabilitation: Human Kinetics Publishers.
Mcgill, S. & Brown, S., 1992. Creep response of the lumbar spine to prolonged full flexion. Clinical Biomechanics, 7 (1), 43-46.
Mcgill, S.M., 1991. Electromyographic activity of the abdominal and low back musculature during the generation of isometric and dynamic axial trunk torque: Implications for lumbar mechanics. Journal of Orthopaedic Research, 9 (1), 91-103.
Miller, E.M., Bazrgari, B., Nussbaum, M.A. & Madigan, M.L., 2012. Effects of gender, preload, and trunk angle on intrinsic trunk stiffness. Journal of Musculoskeletal Research, 15 (02).
Moorhouse, K.M. & Granata, K.P., 2007. Role of reflex dynamics in spinal stability: Intrinsic muscle stiffness alone is insufficient for stability. Journal of Biomechanics, 40 (5), 1058-1065.
Olson, M.W., Li, L. & Solomonow, M., 2004. Flexion-relaxation response to cyclic lumbar flexion. Clinical Biomechanics, 19 (8), 769-776.
Olson, M.W., Li, L. & Solomonow, M., 2009. Interaction of viscoelastic tissue compliance with lumbar muscles during passive cyclic flexion–extension. Journal of Electromyography and Kinesiology, 19 (1), 30-38.
Panjabi, M.M., 1992. The stabilizing system of the spine. Part i. Function, dysfunction, adaptation, and enhancement. Journal of Spinal Disorders, 5, 383-383.
Panjabi, M.M., 2003. Clinical spinal instability and low back pain. Journal of Electromyography and Kinesiology, 13 (4), 371-379.

Parkinson, R.J., Beach, T.a.C. & Callaghan, J.P., 2004. The time-varying response of the in vivo lumbar spine to dynamic repetitive flexion. Clinical Biomechanics, 19 (4), 330-336.
Prado-Leon, L.R., Celis, A. & Avila-Chaurand, R., 2005. Occupational lifting tasks as a risk factor in low back pain: A case-control study in a mexican population. Work, 25 (2), 107.
Punnett, L., Fine, L.J., Keyserling, W.M., Herrin, G.D. & Chaffin, D.B., 1991. Back disorders and nonneutral trunk postures of automobile assembly workers. Scandinavian Journal of Work, Environment and Health, 17 (5), 337.
Rosenbaum, D. & Hennig, E.M., 1995. The influence of stretching and warm-up exercises on achilles tendon reflex activity. Journal of Sports Sciences, 13 (6), 481-490.
Roy, S.H., De Luca, C.J. & Casavant, D.A., 1989. Lumbar muscle fatigue and chronic lower back pain. Spine, 14 (9), 992.
Sbriccoli, P., Solomonow, M., Zhou, B.H. & Lu, Y., 2007. Work to rest durations ratios exceeding unity are a risk factor for low back disorder; a feline model. Journal of Electromyography and Kinesiology, 17 (2), 142-152.
Shin, G. & D’souza, C., 2010. Emg activity of low back extensor muscles during cyclic flexion/extension. Journal of Electromyography and Kinesiology, 20 (4), 742-749.
Shirazi-Adl, A., 2006. Analysis of large compression loads on lumbar spine in flexion and in torsion using a novel wrapping element. Journal of Biomechanics, 39 (2), 267-275.
Solomonow, M., 2011. Neuromuscular manifestations of viscoelastic tissue degradation following high and low risk repetitive lumbar flexion. Journal of Electromyography and Kinesiology.
Solomonow, M., Zhou, B.H., Baratta, R., Lu, Y. & Harris, M., 1999. Biomechanics of increased exposure to lumbar injury caused by cyclic loading: Part 1. Loss of reflexive muscular stabilization. Spine, 24 (23), 2426.
Stobbe, T.J., Plummer, R.W., Jensen, R.C. & Attfield, M.D., 1988. Incidence of low back injuries among nursing personnel as a function of patient lifting frequency. Journal of Safety Research, 19 (1), 21-28.
Taylor, D.C., Dalton, J.D., Seaber, A.V. & Garrett, W.E., 1990. Viscoelastic properties of muscle-tendon units. The American Journal of Sports Medicine, 18 (3), 300-309.
Youssef, J., Davidson, B., Zhou, B.H., Lu, Y., Patel, V. & Solomonow, M., 2008. Neuromuscular neutral zones response to static lumbar flexion: Muscular stability compensator. Clinical Biomechanics, 23 (7), 870-880.
Zhang, L.Q., Huang, H., Sliwa, J.A. & Rymer, W.Z., 1999. System identification of tendon reflex dynamics. Rehabilitation Engineering, IEEE Transactions on, 7 (2), 193-203.

7. Supplemental Studies

In addition to the studies summarized above, which were included in the formal proposal and conducted to address the specific aims, several other studies were completed by the current investigators on related topics. Those that were supported by grant funding, either directly or indirectly, and for which the research is completed, are summarized in abstract form below.

6.1 Trunk Dynamic Responses to Sudden Perturbations

Bazrgari B, Nussbaum MA, Madigan ML, Shirzi-Adl A: [2011] Soft tissue wobbling affects trunk dynamic response in sudden perturbations. Journal of Biomechanics 44:547-551.

Soft tissue wobbling reduces the transferred impact of external loads on lower limb joints. The present study investigated whether soft tissue wobbling has similar effects on the trunk dynamic response to sudden perturbations. Three healthy males were subjected to a series of anteriorly-directed trunk position perturbations at three different velocities while trunk kinematics and kinetics were measured. A nonlinear active-passive finite element model of the human trunk was then used to study the effects of soft tissue wobbling on trunk response. Also investigated were the effects on model predictions of including elements simulating the apparatus (rod-harness assembly) transferring motor-generated perturbations to the trunk. Predicted and measured trunk kinematics and kinetics, when accounting for the dynamic effects of both wobbling mass and rod-harness assembly, were in good agreement for all velocities especially early (< 120 ms) after the perturbations ($\rho > 0.97$). Root mean square errors in model predictions increased considerably when neglecting the aforementioned modeling considerations. The trunk wobbling mass and connecting elements between the trunk and the perturbing device, particularly during faster perturbations, substantially attenuated the transferred impact of external loads on the spine (by 33 – 90 N across perturbation velocities). Such reductions in the impacts transferred, in turn, reduce the predicted demands on the neuromuscular system for control and maintenance of spinal loads and stability. As such, these features should be considered in future biodynamic models of the human trunk aimed at estimating trunk neuromuscular behaviors during sudden perturbations.

6.2 Assumptions in Estimating Trunk Mechanical Properties

Bazrgari B, Nussbaum MA, Madigan ML: [2012] Estimation of trunk mechanical properties using system identification: effects of experimental setup and modelling assumptions. Computer Methods in Biomechanics and Biomedical Engineering 15:1001-1009.

The use of system identification to quantify trunk mechanical properties is growing in biomechanics research. The effects of several experimental and modelling factors involved in the system identification of trunk mechanical properties were investigated. Trunk kinematics and kinetics were measured in six individuals when exposed to sudden trunk perturbations. Effects of motion sensor positioning and properties of elements between the perturbing device and the trunk were investigated by adopting different models for system identification. Results showed that by measuring trunk kinematics at a location other than the trunk surface, the deformation of soft tissues are erroneously included into trunk kinematics and result in the trunk being predicted as a more damped structure. Results also showed that including elements between the trunk and the perturbing device in the system identification model did not substantially alter model predictions. Other important parameters that were found to substantially affect predictions were the cut-off frequency used when low-pass filtering raw data and the data window length used to estimate trunk properties.

6.3 Reliability of Measures of Neuromuscular Behaviors

Hendershot BD, Bazrgari B, Nussbaum MA, Madigan ML: [2012] Within- and between-day reliability of trunk mechanical behaviors estimated using position-controlled perturbations. Journal of Biomechanics 45:2019-2022.

Recent applications of position-controlled perturbation techniques to the human trunk have allowed separate estimation of intrinsic and reflexive trunk mechanical behaviors. These mechanical behaviors play an important role in spinal stability and have been associated with low back pain risk, yet the reliability of these measures remains unknown. Therefore, the objective of the current study was to assess within- and between-day reliability of several measures of trunk mechanical behaviors obtained from position-controlled trunk perturbations. A secondary objective was to assess if different harness designs, used to connect a participant to the perturbing device, influenced reliability. Data were analyzed from baseline measurements obtained from two previously published studies, and a third unpublished study. The total combined subject pool included 33 healthy young adults (17 M, 16 F). Relative and absolute reliability was quantified using intraclass correlation coefficients (ICCs) and standard errors of measurement (SEM), respectively. Within-day ICCs of intrinsic trunk stiffness (0.84-0.90) and effective mass (0.91-95) were excellent, and were generally higher than ICCs for reflex gain (0.55-0.85), maximum reflex force (0.65-0.85), and timing of maximum reflex force (0.48-0.86). Within-day ICCs (0.48-0.95) were consistently superior to between-day values (0.19-0.72). Improvements in harness design increased both within- and between-day reliability and reduced SEMs for most measures.

6.4 Effects of Gender and Task Conditions on Trunk Stiffness Measures

Miller EM, Bazrgari B, Nussbaum MA, Madigan ML: [2012] Effects of gender, preload, and trunk angle on intrinsic trunk stiffness. Journal of Musculoskeletal Research 15:1250012 (9 pages).

Gender, lifting loads, and flexed trunk postures are three risk factors associated with low back pain. Previous studies have not found gender differences in effective trunk stiffness (intrinsic stiffness plus reflex response) using force perturbations, but these measures may have been confounded by differences in trunk kinematics between males and females. The purpose of this study was to investigate the effects of gender, trunk extensor preload, and trunk flexion angle on intrinsic trunk stiffness using position perturbations, which have the potential to eliminate kinematic differences between research subjects and to separate intrinsic stiffness from reflex responses. Thirteen males and twelve females were exposed to sudden, small trunk flexion position perturbations with two trunk extension preloads (0% and 30% maximum) and three trunk flexion angles (0, 20, and 40 degrees). Data collected during position perturbations were used along with a two degree of freedom model of the trunk and connecting elements to estimate trunk intrinsic stiffness. Intrinsic stiffness was lower in females compared to males, and increased with increasing preload and trunk flexion angle. Intrinsic stiffness increased more substantially among males with increasing preload and trunk angle, and effects of trunk angle were diminished with a preload. A lower intrinsic stiffness and smaller increases with preload and trunk angle, may contribute to the increased rate of occupational LBP and injury among females.

6.5 Measuring and Modeling Load-Relaxation in the Lumbar Spine

Toosizadeh N, Nussbaum MA, Bazrgari B, Madigan ML: [2012] Load-relaxation properties of the human trunk in response to prolonged flexion: measuring and modeling the effect of flexion angle. PLoS ONE, Accepted.

Experimental studies suggest that prolonged trunk flexion reduces passive support of the spine. To understand alterations of the synergy between active and passive tissues following such loadings, several studies have assessed the time-dependent behavior of passive tissues including those within spinal motion segments and muscles. Yet, there remain limitations regarding load-relaxation of the lumbar spine in response to flexion exposures and the influence of different flexion angles. Ten healthy participants were exposed for 16 min to each of five magnitudes of lumbar flexion specified relative to individual flexion-relaxation angles (i.e., 30, 40, 60, 80, and 100%), during which lumbar flexion angle and trunk moment were recorded. Outcome measures were initial trunk moment, moment drop, parameters of four viscoelastic models (i.e., Standard Linear Solid model, the Prony Series, Schapery's Theory, and the Modified Superposition Method), and changes in neutral zone and viscoelastic state following exposure. There were significant effects of flexion angle on initial moment, moment drop, changes in normalized neutral zone, and some parameters of the Standard Linear Solid model. Initial moment, moment drop, and changes in normalized neutral zone increased exponentially with flexion angle. Kelvin-solid models produced better predictions of temporal behaviors. Observed responses to trunk flexion suggest nonlinearity in viscoelastic properties, and which likely reflected viscoelastic behaviors of spinal (lumbar) motion segments. Flexion-induced changes in viscous properties and neutral zone imply an increase in internal loads and perhaps increased risk of low back disorders. Kelvin-solid models, especially the Prony Series model appeared to be more effective at modeling load-relaxation of the trunk.

6.6 The Influence of Load Relaxation on Spine Loads during a Subsequent Lift

Toosizadeh N, Nussbaum MA: [nd] Prolonged trunk flexion can increase spine loads during a subsequent lifting task: an investigation of the effects of trunk flexion duration and angle using a viscoelastic spine model. Journal of Biomechanics, Submitted.

Load-relaxation of the human trunk following prolonged flexion has been observed earlier, yet the adverse effects of such viscoelastic behaviors on performing demanding tasks (e.g., lifting) remain poorly understood. Theoretically, flexion exposures reduce trunk stiffness and yield a compensatory increase in paraspinal muscle activation and spine loads. Here, a multi-segment model with nonlinear viscoelastic properties was developed. Viscoelastic material properties were defined using SNS components, and a kinematics-driven approach in combination with an optimization algorithm was used to estimate muscle forces and spine loads during simulated lifting tasks. The model was calibrated and evaluated using previously reported data. Good agreement with existing *in vivo* and modeling reports was found both for load-relaxation responses to prolonged trunk flexion and spine load estimations during lifting. Subsequently, the model was used to predict changes, resulting from a range of trunk flexion exposures, in several outcome measures (i.e., peak spine load, peak axial stiffness, and absorbed energy) at L5/S1 during simulated lifting. All three measures increased following flexion exposures, and these changes were magnified by increasing flexion duration and angle. Increases in spine loads were up to ~9% (~284N), and were primarily a consequence of an increased contribution of active muscle components required to complete a lifting task following the most extreme load-relaxation exposure. These results support prior epidemiological evidence that occupational low back injury risk is elevated when prolonged trunk flexion along with lifting are required.

LIST OF PUBLICATIONS

The following lists publications that have resulted to date from work supported (directly or indirectly) by this grant, and a list of future publications that are anticipated. For each paper, a brief summary is noted, as is the contribution to each of the four Specific Aims.

Journal Articles

1. Bazrgari B, Hendershot B, Muslim K, Toosizadeh N, Nussbaum MA, Madigan ML: [2011] Disturbance and recovery of trunk mechanical and neuromuscular behaviors following prolonged trunk flexion: influences of duration and external load on creep-induced effects. Ergonomics 54:1043-1052

   There are adverse effects of prolonged trunk flexion, and these effects are modified by the duration of flexion and somewhat by external loads. [Specific Aim 1]

2. Hendershot B, Bazrgari B, Muslim K, Toosizadeh N, Nussbaum MA, Madigan ML: [2011] Disturbance and recovery of trunk stiffness and reflexive muscle responses following prolonged trunk flexion: influences of flexion angle and duration. Clinical Biomechanics 26:250-256.

   Exposure to prolonged trunk flexion changed trunk stiffness and reflex behavior in patterns consistent with epidemiological evidence linking such exposure with the risk of occupational low back disorders. [Specific Aim 2]

3. Bazrgari B, Nussbaum MA, Madigan ML, Shirzi-Adl A: [2011] Soft tissue wobbling affects trunk dynamic response in sudden perturbations. Journal of Biomechanics 44:547-551.

   The trunk wobbling mass and connecting elements between the trunk and the perturbing device, particularly during faster perturbations, substantially attenuate the transferred impact of external loads on the spine. Such reductions in the impacts transferred, in turn, reduce the predicted demands on the neuromuscular system for control and maintenance of spinal loads and stability. As such, these features should be considered in biodynamic models of the human trunk aimed at estimating trunk neuromuscular behaviors during sudden perturbations. [Methods in Specific Aim 1, 2, 3, and 4]

4. Bazrgari B, Nussbaum MA, Madigan ML: [2012] Estimation of trunk mechanical properties using system identification: effects of experimental setup and modelling assumptions. Computer Methods in Biomechanics and Biomedical Engineering 15:1001-1009.

   By measuring trunk kinematics at a location other than the trunk surface, the deformation of soft tissues are erroneously included into trunk kinematics and result in the trunk being predicted as a more damped structure. Including elements between the trunk and the perturbing device in the system identification model did not substantially alter model predictions. Other important parameters that were found to substantially affect predictions were the cut-off frequency used when low-pass filtering raw data and the data window length used to estimate trunk properties. [Methods in Specific Aims 1, 2, 3, 4]

5. Hendershot BD, Bazrgari B, Nussbaum MA, Madigan ML: [2012] Within- and between-day reliability of trunk mechanical behaviors estimated using position-controlled perturbations.

Journal of Biomechanics 45:2019-2022.

Within-day reliability of intrinsic trunk stiffness and effective mass were excellent, and were generally higher than levels for reflex gain, maximum reflex force, and timing of maximum reflex force. Within-day reliability was consistently superior to between-day levels. Improvements in harness design increased both within- and between-day reliability and reduced standard errors-of-measurement for most measures. [Methods in Specific Aims 1, 2, 3, and 4]

6. Miller EM, Bazrgari B, Nussbaum MA, Madigan ML: [2012] Effects of gender, preload, and trunk angle on intrinsic trunk stiffness. Journal of Musculoskeletal Research 15:1250012 (9 pages).

   Intrinsic stiffness was lower in females compared to males, and increased with increasing preload and trunk flexion angle. Intrinsic stiffness increased more substantially among males with increasing preload and trunk angle, and effects of trunk angle were diminished with a preload. A lower intrinsic stiffness and smaller increases with preload and trunk angle, may contribute to the increased rate of occupational LBP and injury among females. [Methods and Interpretation of Specific Aims 1, 2, 3, and 4]

7. Toosizadeh N, Nussbaum MA, Bazrgari B, Madigan ML: [2012] Load-relaxation properties of the human trunk in response to prolonged flexion: measuring and modeling the effect of flexion angle. PLoS ONE, Accepted.

   Flexion-induced changes in viscous properties and neutral zone imply an increase in internal loads and perhaps increased risk of low back disorders. Kelvin-solid models, especially the Prony Series model appeared to be more effective at modeling load-relaxation of the trunk. [Methods and Interpretation of Specific Aim 2]

Conference Proceedings

1. Bazrgari B., Nussbaum MA, Madigan ML: [2010] Effects of experimental setup and modeling assumptions on predicted trunk properties using a system identification method (poster). Proceedings of the American Society of Biomechanics Annual Meeting. Providence, RI. August 18-21. Online only, not paginated (2 pp.).
2. Hendershot B, Bazrgari B, Muslim K, Toosizadeh N, Nussbaum MA, Madigan ML: [2010] Disturbances to intrinsic stiffness and reflexive muscle responses following prolonged trunk flexion (poster). Proceedings of the American Society of Biomechanics Annual Meeting. Providence, RI. August 18-21. Online only, not paginated (2 pp.).
3. Miller E, Bazrgari B, Hendershot B, Nussbaum MA, Madigan ML: [2010] Dynamic response of the trunk to position perturbations: effects of gender, preload, and trunk angle (poster). Proceedings of the American Society of Biomechanics Annual Meeting. Providence, RI. August 18-21. Online only, not paginated (2 pp.).
4. Toosizadeh N, Bazrgari B, Hendershot B, Muslim K, Nussbaum MA: [2010] In vivo Load-relaxation of the trunk with prolonged flexion (podium). Proceedings of the American Society of Biomechanics Annual Meeting. Providence, RI. August 18-21. Online only, not paginated (2 pp.).
5. Miller E, Bazrgari B, Nussbaum M, Madigan M: [2011] Effects of exercise-induced low back pain on intrinsic trunk stiffness. Proceedings of the American Society of Biomechanics Annual Meeting. Long Beach, CA. August 10 – 13. Online only, not paginated (2 pp.).

6. Hendershot B, Bazrgari B, Nussbaum MA, Madigan M: [2011] Comparison of mechanical- and EMG-based estimates of trunk reflexes to sudden perturbation. Proceedings of the Biomedical Engineering Society Annual Meeting. Hartford, CT. October 12-15. Abstract only.
7. Bazrgari B, Nussbaum MA, Madigan ML, Shirazi-Adl A: [2012] Trunk dynamic responses to sudden perturbations: Effects of soft tissue wobbling. Proceedings of the 4th American Conference on Human Vibration, Hartford, CT. June 13-15. pp. 113-114.
8. Hendershot BD, Nussbaum MA: [2012] Reduced and asymmetric trunk stiffness among unilateral lower-limb amputees during multi-directional trunk perturbations. Presented at the American Society of Biomechanics Annual Meeting. Gainesville, FL. August 15-18. Online only, not paginated (2 pp.).
9. Muslim K, Hendershot B, Bazrgari B, Toosizadeh N, Nussbaum MA, Madigan ML: [2012] Disturbances to intrinsic stiffness and reflexive muscles responses following repeated static trunk flexion. Presented at the American Society of Biomechanics Annual Meeting. Gainesville, FL. August 15-18. Online only, not paginated (2 pp.).
10. Toosizadeh N, Nussbaum MA, Madigan ML: [2012] Viscoelastic modeling of the lumbar spine: the effect of prolonged flexion on internal loads. Presented at the American Society of Biomechanics Annual Meeting. Gainesville, FL. August 15-18. Online only, not paginated (2 pp.).

Dissertation/Thesis

1. Hendershot B: [2012] Alterations and asymmetries in trunk mechanics and neuromuscular control among persons with lower-limb amputation: exploring potential pathways of low back pain, PhD Thesis, Virginia Tech.
2. Miller E: [2012] Exercise-induced low back pain and neuromuscular control of the spine: experimentation and simulation, PhD Thesis, Virginia Tech.
3. Toosizadeh N: [expected early 2013] Time-dependent assess of the human lumbar spine in response to flexion exposures: *in vivo* measurement and mdoeling, PhD Thesis, Virginia Tech.

Publications Submitted or In Preparation

1. Muslim K, Bazrgari B, Hendershot B, Toosizadeh N, Nussbaum MA, Madigan ML: [2012] Disturbance and recovery of trunk mechanical and neuromuscular behaviors following repeated static trunk flexion: influences of duration and duty cycle on creep-induced effect. Applied Ergonomics, Revision In Progress

   Reflexive responses may not provide a compensatory mechanism to offset decreases in intrinsic trunk stiffness following repetitive static trunk flexion. A prolonged recovery duration may lead to trunk instability and a higher risk of low back injury. [Specific Aim 3]

2. Toosizadeh N, Bazrgari B, Hendershot B, Muslim K, Nussbaum MA, Madigan ML: [ND] Disturbance and recovery of trunk mechanical and neuromuscular behaviors following repetitive lifting: influences of flexion angle and lift rate on creep-induced effect. Ergonomics, In Preparation.

   Intrinsic trunk stiffness and reflexive responses were compromised following lifting exposures, with larger decreases in stiffness and reflexive force caused by larger flexion angles. Simultaneous changes in both trunk stiffness and neuromuscular behaviors may *impose an increased risk of trunk instability and low back injury. [Specific Aim 4]*

3. Toosizadeh N, Nussbaum MA: [nd] Prolonged trunk flexion can increase spine loads during a subsequent lifting task: an investigation of the effects of trunk flexion duration and angle using a viscoelastic spine model. Journal of Biomechanics, Submitted.

   Peak lumbosacral load, peak axial stiffness, and absorbed energy during simulated lifting all increased following flexion exposures, and these changes were magnified by increasing flexion duration and angle. Increases in spine loads were up to ~9% (~284N), and were primarily a consequence of an increased contribution of active muscle components required to complete a lifting task following the most extreme load-relaxation exposure. These results support prior epidemiological evidence that occupational low back injury risk is elevated when prolonged trunk flexion along with lifting are required. [Interpretation of Specific Aim 4]

1. Wojcik LA, Shibata PA, Nussbaum MA, Lin D, Madigan ML: [Revision in Preparation] Age and gender differences in the effects of localized muscle fatigue on lower extremity joint torques used during quiet stance. Journal of Applied Biomechanics.

   An inverse dynamics model was developed and used to assess the involvement of major joints in the control an upright posture, as well as the influences of age and fatigue. [Specific Aim 3]

INCLUSION OF GENDER AND MINORITY STUDY SUBJECTS

Study Title: Occupational Trunk Flexion and Neuromuscular Disturbance
Total Enrollment: 58
Grant Number: R01 OH08504

PART A. TOTAL ENROLLMENT REPORT: Number of Subjects Enrolled to Date (Cumulative) by Ethnicity and Race

Ethnic Category	Females	Males	Unknown or Not Reported	Total
Hispanic or Latino	1	1	0	2 **
Not Hispanic or Latino	27	29	0	56
Unknown (individuals not reporting ethnicity)	0	0	0	0
Ethnic Category: Total of All Subjects*	28	30	0	58 *
Racial Categories				
American Indian/Alaska Native	0	0	0	0
Asian	1	2	0	3
Native Hawaiian or Other Pacific Islander	0	0	0	0
Black or African American	2	2	0	4
White	24	25	0	49
More Than One Race	1	1	0	2
Unknown or Not Reported	0	0	0	0
Racial Categories: Total of All Subjects*	28	30	0	58 *

PART B. HISPANIC ENROLLMENT REPORT: Number of Hispanics or Latinos Enrolled to Date (Cumulative)

Racial Categories	Females	Males	Unknown or Not Reported	Total
American Indian or Alaska Native	0	0	0	0
Asian	0	0	0	0
Native Hawaiian or Other Pacific Islander	0	0	0	0
Black or African American	0	0	0	0
White	1	1	0	2
More Than One Race	0	0	0	0
Unknown or Not Reported	0	0	0	0
Racial Categories: Total of Hispanics or Latinos**	1	1	0	2 **

INCLUSION OF CHILDREN

Most of the studies conducted under this grant involved children (i.e. those 18-21 years of age). Many workers are children by this standard, and are exposed to conditions of interest here, specifically where there prolonged and/or repetitive trunk flexion (or lifting). Hence, the results of the research described here have relevance for conditions affecting children.

MATERIALS AVAILABLE FOR OTHER INVESTIGATORS

All experimental data are available. Software (MatLab™ code) is available for the biomechanical models. These may be accessed by contacting the PI, whose information is provided below. Note that the investigators wish to limit such access to individuals requesting data or software for research purposes only.

Maury A. Nussbaum, PhD
Virginia Tech
250 Durham Hall (0118)
Blacksburg, VA 24061
USA
(540) 231-6053
nussbaum@vt.edu