Risk of Lumbar Spine Injury from Cyclic Compressive Loading

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Study Design: Survival analyses of a large cohort of published lumbar spine compression fatigue tests.
Objective: To produce the first large-scale evaluation of human lumbar spine tolerance to repetitive compressive forcing and to evaluate and improve guidelines for human exposure to whole body vibration and repeated mechanical shock environments.
Summary of Background Data: Several studies have examined the effects of compressive cyclic loading on the lumbar spine. However, no previous effort has coalesced these studies and produced an injury risk analysis with an expanded sample size. Guidelines have been developed for exposure limits to repetitive loading, (e.g., ISO 2631-5,) but there has been no large scale verification of the standard against experimental data.
Methods: Survival analyses were performed using the results of 78 male and 29 female cadaveric spinal segment fatigue tests from six previously published studies. Segments were fixed at each end and exposed to pure axial, cyclic compression. The effects of the number of cycles, load amplitude, sex, and age were examined through the use of survival analyses.
Results: Number of cycles, load amplitude, sex, and age all are significant factors in the likelihood of bony failure in the spinal column. Using a modification of the risk prediction parameter from ISO 2631-5, an injury risk model was developed which relates risk of vertebral failure to repeated compressive loading. The model predicts lifetime risks less than 5% for industrial machinery exposure from axial compression alone. A risk of 35% was found for a high speed planing craft operator, consistent with epidemiological evidence.
Conclusions: A spinal fatigue model has been developed which predicts the risk of *in vitro* lumbar spinal failure within a narrow confidence interval. Age and sex were found to have significant effects on fatigue strength, with sex differences extending beyond those accounted for by endplate area disparities.

Key Points:

- This study provides the first repeated axial compression injury risk criteria for lumbar spinal bony injury in terms of peak lumbar spine stress, age, and number of loading cycles and includes the effects of sex.
- The model includes the effect of sex on lumbar fatigue strength and reveals sex differences which extend beyond those accounted for by endplate area disparities.
- The model predicts lifetime risks less than 5% for industrial machinery exposure from axial compression alone. A risk of 35% was found for a high speed planing craft operator, consistent with epidemiological evidence.

INTRODUCTION:

Lumbar spinal injuries and associated chronic back pain cause widespread morbidity, and exposure to cyclic vertical loading and whole body vibration is suspected to be a contributing factor[1-3]. Extreme sources of such repeated loading include high speed planing watercraft[4,5] and high performance aircraft[6,7]. Potential pathologies from repeated lumbar spinal stress may present clinically in many forms and reflect a range of etiologies from simple muscle strain to severe degenerative lesions, disk herniation, or acute fracture at higher force levels[8-11]. The presence of signs and symptoms may lead to further work-up, including radiographic imaging, but resultant tissue imaging findings may be elusive or difficult to interpret diagnostically[12].

Human studies on the association of injury risk with mechanical predictor variables are complicated by the high incidence of preexisting lumbar spinal abnormalities (including disc herniation) in asymptomatic individuals with no apparent history of spinal trauma[13]. In response, many *in vitro* studies have been conducted using cadaveric and animal functional spinal units to assess the effects of repetitive loading on the spine[11,14-18].

For compressive axial loading, these *in vitro* studies were typically performed under load control; this is similar to the expected human seated or standing loading profile in which functional spinal units creep under cyclic loading with similar cycle to cycle peak loads. Hansson focused on the influence of functional spinal stiffness and fracture morphology[16] while Brinckmann[11] and Huber[18] reported a correlation of peak axial force to failure with number of loading cycles[20-22]. Previous studies have generally found age[16,18] and endplate area[11,19] to have a qualitative effect on lumbar spinal injury in repeated loading. Though Brinckmann et al [11] did not find an age effect, this was likely the result of small sample sizes and censoring caused by specimen selection. However, these types of *in vitro* models have not typically produced herniation in pure axial loading in the absence of fractures or avulsions. Since the human spine is adapted to the repeated loading of bipedal walking and running[20], initial and dynamic posture affect intervertebral loading and injury incidence[21,22], and herniation is generally associated with cyclic flexion/extension and other bending/torsion modes applied with relatively low magnitude compressive loading[14,23].

Several biomechanical risk assessments have established limits on mechanical loading for personnel exposed to single and repeated axial compressive loading[24-27]. The current ISO standard 2631-5:2004 provides guidance on the health effects of exposure to repeated mechanical shocks using a parameter termed the R value[26]. The R value is based on an accelerated failure model for the lumbar spine[28] and is proportional to the maximum static stress per cycle, the sixth root of the number of cycles, and inversely proportional to the static ultimate strength of a spinal segment. The formulation reduces to $R = S_{max} C^{\frac{1}{6}} / (S_{ui} - g)$, where S_{Max} is the maximum stress during a cycle, C is the number of cycles, S_{ui} is the static ultimate strength of the spinal segment, and g represents the static stress due to gravitational force for a given posture (the standard suggests 0.25 MPa for the driving position.) The ultimate strength of the spine is estimated as $S_{ui} = 6.75 - G_{age} \cdot Age$, where G_{age} is an age-dependence coefficient with a value of 0.066. However, the value assigned G_{age} was developed based on single-cycle compression tests[26] and has not previously been validated experimentally for cyclic loading. The standard defines lifetime cumulative R values greater than 0.8 as moderate risk for injury, and 1.2 is given as a high risk, though the nature of this injury risk is not further specified.

There is no current injury criterion that quantifies the risk of lumbar spine endplate fracture or other spinal repeated loading fracture from axial compressive loading in terms of peak loading, cycles to failure, age, and sex. This study uses male and female cadaveric functional spinal unit tests in the literature to assess the age-dependent risk of injury in cyclic loading.

MATERIALS AND METHODS

An analysis of lumbar spinal injury risk was performed using the results of 107 cadaveric spine fatigue tests from six studies on cyclic axial loading injury[11,14-18]. The donors were aged 19 to 93 years at time of death, and included 78 male and 29 female specimens. The age distributions were relatively uniform, with a mean age for males of 53 ± 19 years and 59 ± 20 years for females. Each specimen was designated by the original author as representative of a normal patient, free of substantial dysfunction or spinal trauma. In each of these tests, the specimen was a lumbar spinal segment composed of two vertebral bodies and the intervertebral disc with posterior elements and ligaments intact; segments considered for inclusion were between T12-L1 and L5-S1. Each segment was fixed at both ends and exposed to pure axial, cyclic compression at loads between 400 and 7100 N. Exposures ranged in frequency from 0.25 Hz to 5 Hz for a maximum of 1.29×10^6 cycles with a median of 1,900 cycles. Determination of injury was made by the original authors; in the cases included in this analysis, all injuries were classified as damage to the endplate or general bony structure.

The current analyses were conducted using the age and sex of the donor, the number of cycles to which the specimen was exposed, and the effective stress applied over the endplate area during each cycle. Effective stress was used instead of peak axial load for two reasons. First, the use of stress provided a more robust comparison between samples at different vertebral levels and from cadavers of different sizes. Second, existing injury criteria and exposure limits for repeated vertebral loading are calculated in terms of stress, not load, so the conversion was necessary for comparison.

The effective applied stress was defined as the load transmitted through the intervertebral disc divided by the endplate area. This definition excluded facet contributions to account for postural effects. Using the findings of Hakim and King among others[29-31], facet contributions were estimated to carry 26% of the load during compression in a typical neutral seated posture. For each trial, the peak load was decremented by that percentage to represent the load borne individually by the intervertebral disc. The decremented load was then divided by the measured vertebral endplate area to yield effective stress. For 39 specimens, the unreported endplate areas were estimated using the average of published endplate areas[18,22] for the appropriate spinal level and sex (Figure 1). It is assumed that the resulting stress sufficiently accounts for the variation in spinal level.

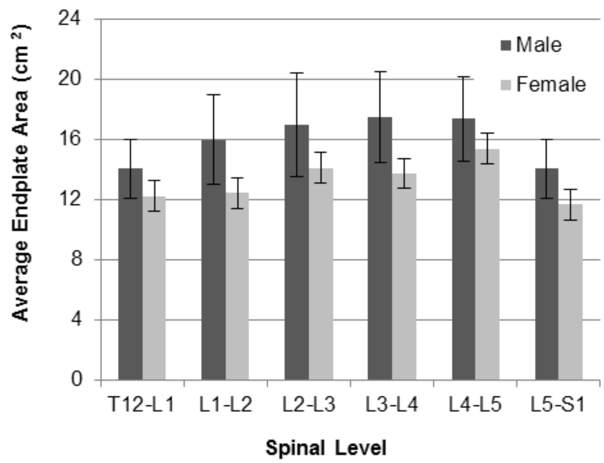

Figure 1. Averaged endplate areas used where specimen dimensions were unreported. Insufficient data was available for the female L5-S1 joint, so that area was estimated by scaling from the male data using the average proportion 0.83 (±0.04).

Two models were developed using the statistical analysis package JMP v. 9.0.1 (SAS Institute, Inc., Cary, NC). The first was a parametric survival analysis where the probability of failure was determined as a function of the number of cycles, stress, and age, with coefficients determined separately for each sex. The model was based on a Weibull distribution of the form $\Pi = 1 - \exp\left[-\left(\frac{C}{a_1}\right)^{b_1}\right]$, where *C* is the number of cycles, b_1 is a constant shape parameter, and $a_1 = \exp(K_0 + K_1 \cdot Age + K_2 \cdot Stress)$.

In the second model, the effects of age, stress, and cycles were combined into a modified version of the ISO 2631 R parameter to quantify the cumulative exposure for a univariate injury risk curve. The minimum stress during each cycle was interpreted as equivalent to the static gravitational stress under physiological seated conditions with an assumed upper body mass. In this intermediate model, R was the time to failure, and age was included as a model effect ($\Pi = 1 - \exp\left[-\left(\frac{R}{a_2}\right)^{b_2}\right]$, $a_2 = \exp(C_0 + C_1 \cdot Age)$). Here, two coefficients described the effects of age: C_1 and the G_{age} coefficient within the R value. For each sex, an optimized value of the G_{age} coefficient was determined by adjusting G_{age} until the model coefficient for age dependence, C_1, was zero. Thus, the entire effect of age was incorporated into the R expression. Using the optimized G_{age} values, a univariate Weibull survival analysis was performed with $\Pi = 1 - \exp\left[-\left(\frac{R}{\alpha}\right)^{\beta}\right]$, where α is a constant scale parameter.

RESULTS

The coefficients for the parametric survival model are given in Table 1, and Figure 2 shows the relationship between effective stress and cycles at the 50% failure point for various ages. For each sex, both stress and age were statistically significant effects. In the male model, stress and age were significant at $p<0.001$; for females, stress was significant at $p<0.001$, and age was significant at $p<0.05$. The logarithmic character of the relationship between stress and cycles illustrates that the applied stress has a stronger influence on the failure probability than the number of cycles. For instance, for a 20 year old male, doubling the applied stress from 1 to 2 MPa decreases the expected 50% failure cycle from 534,000 to 8,600 cycles – a reduction of over 98%.

Table 1. Coefficients for the parametric survival model (Standard error)

Sex	K_0	K_1	K_2	b_1
Male	19.915 (1.625)	-4.128 (0.519)	-0.091 (0.019)	0.465 (0.057)
Female	18.548 (3.457)	-5.0126 (1.138)	-0.075 (0.031)	0.578 (0.099)

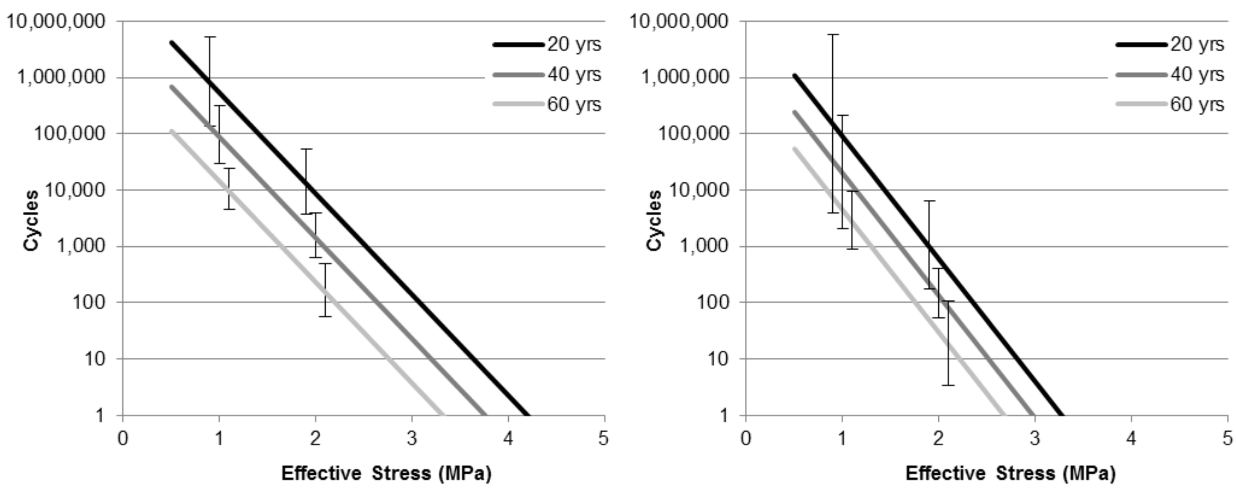

Figure 2. 50% injury risk contours for 20, 40, and 60 year olds with 95% confidence intervals (CI) plotted against a logarithmic scale, emphasizing the stronger effect of stress magnitude than the number of cycles.

The optimized values for G_{age} are reported in Table 2, along with α and β values for the Weibull injury risk curves, shown in Figure 3. The curves demonstrate the substantial discrepancy that exists between male and female dose tolerance – a difference which persists although the loading has been normalized to individual endplate areas. Table 3 reports R values with confidence intervals for specific probabilities of failure.

Table 2. Coefficients for modified R parameter and Weibull injury risk model (95% confidence intervals)

Sex	G_{age}	α	β
Male	0.0524	1.648 (1.504, 1.830)	2.989 (2.346, 3.705)
Female	0.0390	1.014 (0.891, 1.167)	3.228 (3.228, 4.386)

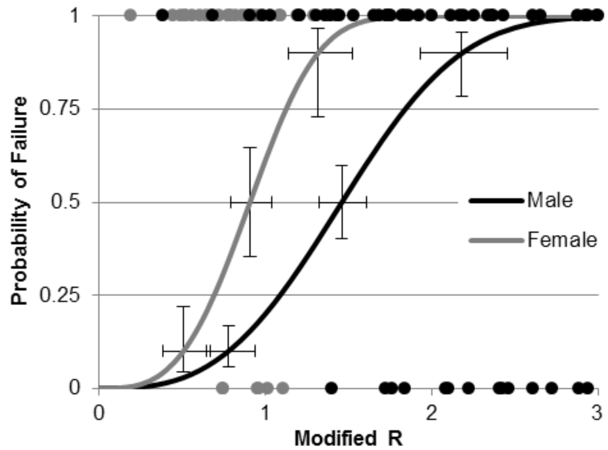

Figure 3. Injury risk curves for males and females with 95% confidence intervals at 0.1, 0.5, and 0.9 probability of failure.

Table 3. Modified R parameters for failure probabilities of 0.1, 0.5, and 0.9

	R-Value (Lower 95% CI, Upper 95% CI)		
	Probability of Failure		
Sex	**10%**	**50%**	**90%**
M	0.78 (0.64, 0.94)	1.46 (1.32, 1.61)	2.18 (1.93, 2.45)
F	0.50 (0.38, 0.66)	0.91 (0.79, 1.04)	1.31 (1.13, 1.52)

Figure 4 illustrates the differences between the modified and unmodified (R_{ISO}) parameters. ISO 2631-5 predicts a "low probability of adverse health effects" for R_{ISO} values less than 0.8, and a high probability for R_{ISO} values greater than 1.2. The curves in Figure 4 were generated by converting those R_{ISO} values to the equivalent modified R value for identical exposure conditions. The comparison highlights both the differences in age dependence and the importance of creating separate models for male and female specimens. While ISO 2631-5 does not quantify what injury rates constitute low and high probability, the original standard tends to underestimate risk for younger adults and greatly overestimate risk for older populations.

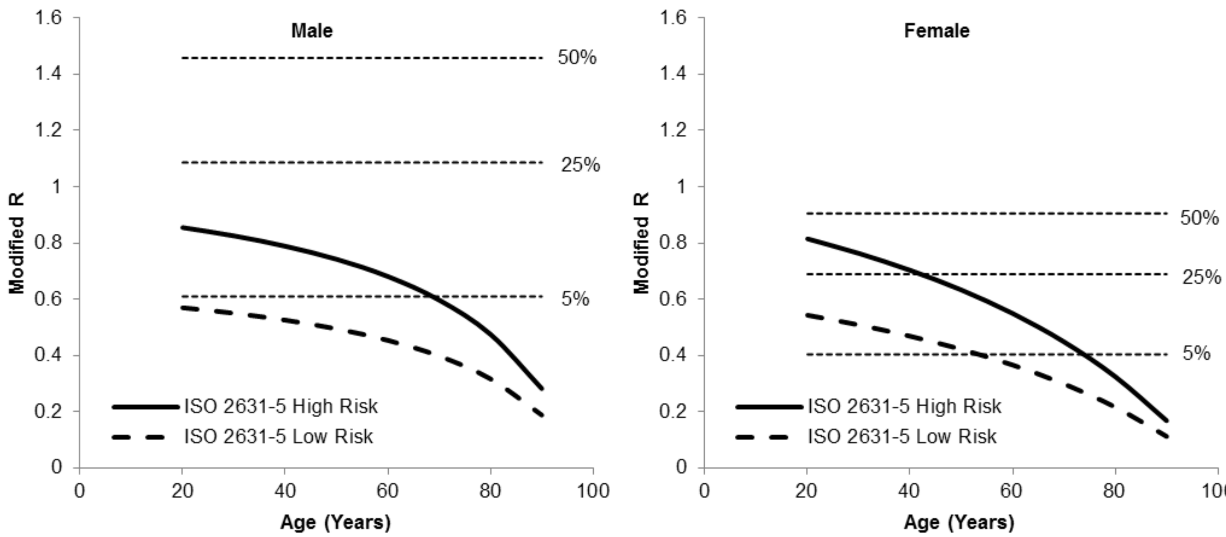

Figure 4. Unmodified ISO 2631-5 injury criteria in terms of the modified R parameter and predicted failure probability from this study. The low risk curves correspond to an unmodified R value of 0.8, and the high risk curves to a value of 1.2.

In the male model, the R_{ISO} = 0.8 contour falls below the 5% failure rate for all ages. The ISO high risk contour for males corresponds to a 13.1% risk of injury at 20 years old, but only a 0.2% risk at age 90. However, the contrast between modified and unmodified parameters is greater for the female model. An exposure that would yield a low-risk R_{ISO} value of 0.8 corresponds to a 12.5% probability of failure for a 20 year old female, and the high-risk limit of R_{ISO}=1.2 corresponds to a failure risk of 39%.

DISCUSSION

This study provides the first repeated axial compression injury risk criterion for lumbar spinal bony injury, including incipient endplate and vertebral body fracture and facet disruption, in terms of peak lumbar spine stress, age, and number of loading cycles and includes the effects of sex. The parametric survival model has statistically significant coefficients and emphasizes that sex influences injury risk in the experimental model. This difference in injury outcome is associated with mechanical characteristics beyond effects from sex-related differences in endplate area alone, which are accounted for using the stress analysis in this study. Previous studies, such as Seidel et al[27] and ISO 2631-5[26], have not assessed this distinction. Interestingly, for all ages, though female specimens can withstand significantly less stress than males, female injury risk curves showed less dependence on age than did the males, and the disparity between the sexes narrows with age (Figure 5).

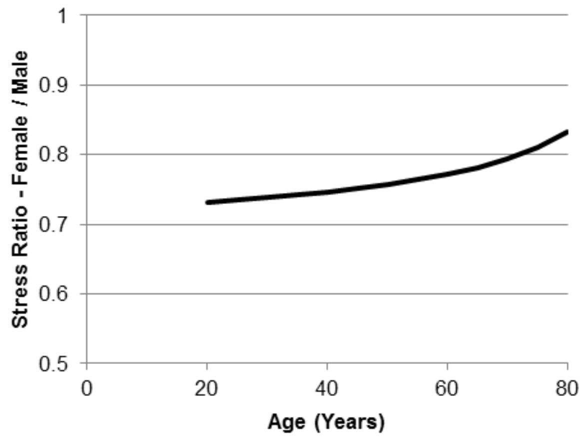

Figure 5. The ratio of applied stress required between female and male vertebrae for a 50% failure rate at 10,000 cycles.

The age effects found in this study are consistent with the effects of declining bone mineral density on compressive strength under repeated loading and ultimate strength[18,19,32], and the age coefficients developed here appear to predict failure more accurately for older specimens than the unmodified ISO standard. For example, the axial repeated loading study of Gallagher et al[17] exposed an aged cohort of 12 cadaveric specimens [average age 80.7 (±7.8)] years to repeated axial compressions of 1,300 N. For those conditions, the R_{ISO} = 1.2 criterion predicted a "high probability of adverse health effects" at 9 cycles; however, the experiment resulted in fracture for only 4 of the 12 specimens after 10,200 cycles and an average endurance of 8,300 (±2,900) cycles until failure. Improved agreement was found by using the modified R formulation in this study, in which the experimental failure rate was predicted as 3100 (-1300,+7500) which overlaps the Gallagher et al experimental values. There may be some influence of selection-based censoring in the experimental models, in that much of the experimental literature uses specimens selected for lack of osteopenia, eliminating samples associated with patients with long bed rest and other factors.

Lifetime risk of injury from occupational repeated loading can be estimated using the model for R developed in this study based on the data shown in Johanning[33] of the root mean squared (RMS) vertical accelerations. Assuming a lifetime of sinusoidal peaks at the reported minimum, mean and maximum values, for a frequency of 1 Hz for 45 years starting at age 20, the risk values shown in Figure 6 are highest for the offroad scraper and forklift truck. Risk values for these machines approach 4% for a lifetime of repeated maximum forcing values reported by Johanning. However, this likely underestimates the risk because RMS accelerations were used in place of full acceleration profiles. Since high acceleration peaks are weighted much more heavily in the exposure parameter, even a very small number of hard jolts would cause a substantial increase. While it has been established that female vertebrae have much lower strength than males, females' calculated injury risks are slightly lower for given acceleration profiles due to the fact that the ratio of upper body mass to lumbar endplate area is, on average, smaller than in males. Injury risk values for the naval Mark V high speed planing craft (HSPC) were reported in Bass et al[4] with exposure assumptions of 20 days/year for a 20 year career. The result predicted high injury risk values of 34.9% (-8.7%,+10.3%), consistent with reported high risk of back injuries in occupants of high speed planing craft[34].

A recent European Union directive[35] limits RMS whole body vibration exposure to an action value of 0.5 m/s^2 and limit value of 1.15 m/s^2. Using the R risk assessment with the same methodology as above, the lifetime risk of injury for a career with the action value is less than 0.1% and the lifetime risk with the limit value is less than 1% (Figure 6).

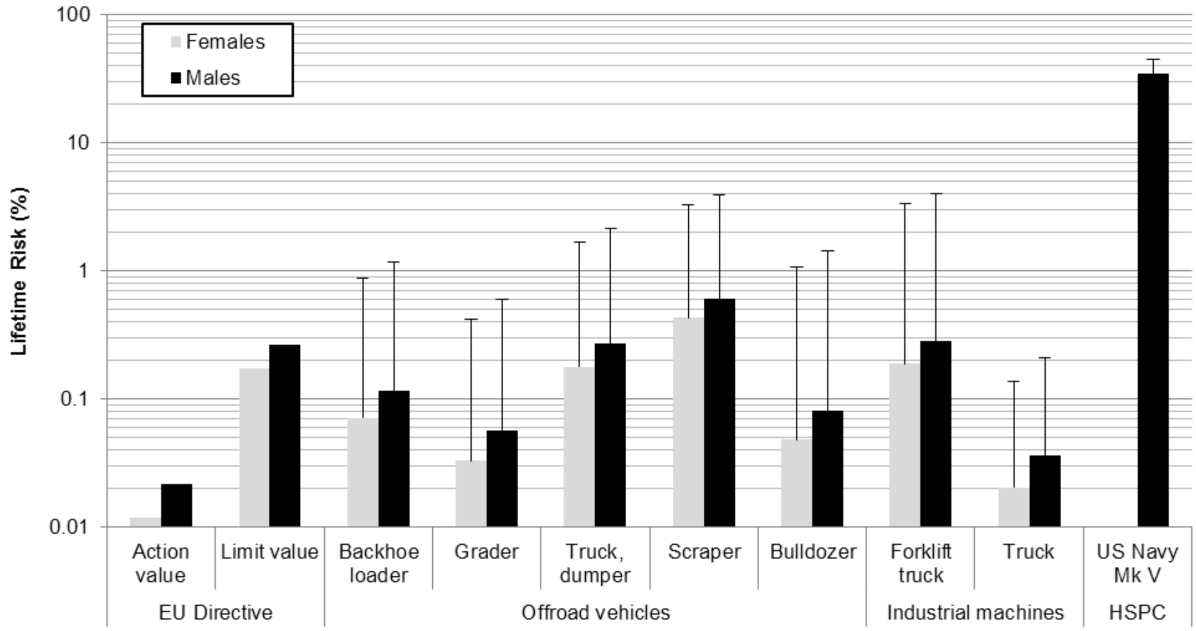

Figure 6. Lifetime risk assessments using modified R based on RMS exposure data for occupations from Johanning (2000)[33] and Bass (2005)[4] compared with EU directive action (0.5 m/s^2 RMS) and limit values (1.15 m/s^2 RMS)[35]. Data bars represent injury risk using mean accelerations; error bars represent risk using maximum peaks.

The Palmgren-Miner power law exponent[28] in the definition of R can be estimated from the risk models assuming a power law relationship between failure cycles and stress and a linear relationship between age and stress that maintains a constant risk between 10 and 10^6 cycles. With this assumption, the power law exponent is 6.3 (R^2=0.94) compared with the current ISO-2631 value of 6.0. Haddock et al found a power law relationship between cycles to failure and stress for human vertebral trabecular bone alone of 9.2[36].

Existing literature has established the load-carrying effects of facets, which act such that vertebral body loading increases during flexion and decreases during extension[29,37], and recent data from a vertebral body implant supports the use of 26% facet contribution in this study[38]. However, alternative load paths between the pelvis and upper torso that are attributable to abdominal musculature and abdominal pressure are difficult to assess and are not included in this model. Measured values of abdominal pressure in humans vary widely, from mean pressures of 0.98 kPa standing quietly[39] to 22.8 kPa while jumping[40]. Furthermore, the overall effect of increased abdominal pressure is unclear. While increased abdominal pressure may help to support the upper body[41,42], it has also been correlated to elevated pressure in the intervertebral disc[43,44], and data from an implanted load cell suggest a limited effect of abdominal load paths in the seated condition[38]. Future work is necessary on contributions from alternative load paths, especially far from the neutral posture.

Limitations of the study include the use of human cadaveric functional spinal units in the injury risk models, with associated differences between *in vivo* end conditions, physiological conditions, musculature effects, and potential differences in accelerated repeated loading testing between *in vitro* and *in vivo* conditions. The effects of flexion and extension superimposed on the axial compressive loading are not included in the models developed in this study, and a neutral posture is assumed for the experimental models. Further, the current study addresses only bony injury, not disc injuries, which can occur at much lower stress levels in non-neutral postures[23]. Since it is known that disc protrusion and subsequent herniation is related to posture[12,21,23,45], further work is necessary to incorporate flexion/extension, torsion and shearing motions. As with the ISO 2631-5 model, the current models include no contribution from healing; this is likely substantial over the course of years.

References:

1. Wilder DG, Pope MH. Epidemiological and aetiological aspects of low back pain in vibration environments--an update. *Clin. Biomech.* 1996;11(2):61-73.

2. Bovenzi M, Hulshof CTJ. An updated review of epidemiologic studies on the relationship between exposure to whole-body vibration and low back pain (1986–1997). *Int. Arch. Occup. Environ. Health.* 1999;72(6):351-365.

3. Seidel H. On the relationship between whole-body vibration exposure and spinal health risk. *Ind. Health.* 2005;43(3):361-377.

4. Bass CR, Salzar R, Ziemba Z, Lucas SR, Peterson R. The modeling and measurement of humans in high speed planing boats under repeated vertical impacts. *International Research Conference on the Biomechanics of Impact (IRCOBI)*. Prague 2005.

5. Gollwitzer R, Peterson R. *Repeated Water Entry Shocks on High-Speed Planing Boats.* Panama City, FL: Naval Surface Warfare Center Dahlgren Division; September 1995.

6. de Oliviera CG, Nadal J. Transmissibility of Helicopter Vibration in the Spines of Pilots in Flight. *Aviat. Space Environ. Med.* 2005;76:576-580.

7. Coakwell MR, Bloswick DS, Moser R. High-Risk Head and Neck Movements at High G and Interventions to Reduce Associated Neck Injury. *Aviat. Space Environ. Med.* 2004;75:68-80.

8. Oxland TR, Panjabi MM. The onset and progression of spinal injury: A demonstration of neutral zone sensitivity. *J. Biomech.* 1992;25(10):1165-1172.

9. Meleger AL, Krivickas LS. Neck and Back Pain: Musculoskeletal Disorders. *Neurol. Clin.* 2007;25(2):419-438.

10. Chou R, Baisden J, Carragee EJ, Resnick DK, Shaffer WO, Loeser JD. Surgery for low back pain: a review of the evidence for an American Pain Society Clinical Practice Guideline. *Spine.* 2009;34(10):1094-1109.

11. Brinckmann P, Biggemann M, Hilweg D. Fatigue Fracture of Human Lumbar Vertebrae. *Clin. Biomech.* 1988;3 (Suppl. 1):1-23.

12. Tampier C, Drake JDM, Callaghan JP, McGill SM. Progressive Disc Herniation: An Investigation of the Mechanism Using Radiologic, Histochemical, and Microscopic Dissection Techniques on a Porcine Model. *Spine.* 2007;32(25):2869-2874.

13. Boden SD, Davis D, Dina T, Patronas N, Wiesel S. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. *The Journal of bone and joint surgery.* 1990;72(3):403.

14. Hardy W, Lissner H, Webster J, Gurdjian E. Repeated Loading Tests of the Lumbar Spine. *Surg. Forum.* 1958;9:690-695.

15. Liu Y, Njus G, Buckwalter J, Wakano K. Fatigue Response of Lumbar Intervertebral Joints Under Axial Cyclic Loading. *Spine.* 1983;8(8):857-865.

16. Hansson TH, Keller TS, Spengler DM. Mechanical behavior of the human lumbar spine. II. Fatigue strength during dynamic compressive loading. *J. Orthop. Res.* 1987;5(4):479-487.

17. Gallagher S, Marras WS, Litsky AS, Burr D. Torso flexion loads and the fatigue failure of human lumbosacral motion segments. *Spine.* 2005;30(20):2265.

18. Huber G, Skrzypiec DM, Klein A, Püschel K, Morlock MM. High Cycle Fatigue Behaviour of Functional Spinal Units. *Ind. Health.* 2010;48(5):550-556.

19. Seidel H, Pöpplau BM, Morlock MM, Püschel K, Huber G. The size of lumbar vertebral endplate areas--Prediction by anthropometric characteristics and significance for fatigue failure due to whole-body vibration. *International Journal of Industrial Ergonomics.* 2007;38(9-10):844-855.

20. Preuschoft H. Mechanisms for the acquisition of habitual bipedality: are there biomechanical reasons for the acquisition of upright bipedal posture? *J. Anat.* 2004;204(5):363-384.

21. Adams MA, Hutton W. Gradual Disc Prolapse. *Spine.* 1985;10(6):524-531.

22. Adams MA, McNally DS, Chinn H, Dolan P. Posture and the compressive strength of the lumbar spine. *Clin. Biomech.* 1994;9(1):5-14.

23. Callaghan JP, McGill SM. Intervertebral disc herniation: studies on a porcine model exposed to highly repetitive flexion/extension motion with compressive force. *Clin. Biomech.* 2001;16:9.

24. Eiband AM. Human tolerance to rapidly applied accelerations: a summary of the literature. *NASA Memo No. 5-19-59E.* National Aeronautics and Space Administration, Washington, DC. 1959.

25. Payne PR. *Personnel Restraint and Support System Dynamics.* Wright-Patterson Air Force Base, Ohio: Aerospace Medical Research Laboratories;1965. AMRL-TR-65_127.

26. International Organization for Standardization. ISO 2631-5:2004 Mechanical vibration and shock -- Evaluation of human exposure to whole-body vibration -- Part 5: Method for evaluation of vibration containing multiple shocks. 2004.