A Comparison of Fatigue Failure Responses of Old *Versus* Middle-Aged Lumbar Motion Segments in Simulated Flexed Lifting

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Study Design. Survival analysis techniques were used to compare the fatigue failure responses of elderly motion segments to a middle-aged sample.

Objectives. To compare fatigue life of a middle-aged sample of lumbosacral motion segments to a previously tested elderly cohort. An additional objective was to evaluate the influence of bone mineral content on cycles to failure.

Summary of Background Data. A previous investigation evaluated fatigue failure responses of 36 elderly lumbosacral motion segments (average age, 81 ± 8 years) subjected to spinal loads estimated when lifting a 9-kg load in 3 torso flexion angles (0°, 22.5°, and 45°). Results demonstrated rapid fatigue failure with increased torso flexion; however, a key limitation of this study was the old age of the specimens.

Methods. Each lumbosacral spine was dissected into 3 motion segments (L1–L2, L3–L4, and L5–S1). Motion segments within each spine were randomly assigned to a spinal loading condition corresponding to lifting 9 kg in 3 torso flexion angles (0°, 22.5°, or 45°). Motion segments were statically loaded and allowed to creep for 15 minutes, then cyclically loaded at 0.33 Hz. Fatigue life was taken as the number of cycles to failure (10 mm displacement after creep loading).

Results. Compared with the older sample of spines, the middle-aged sample exhibited increased fatigue life (cycles to failure) in all the torso flexion conditions. Increased fatigue life of the middle-aged specimens was associated with the increased bone mineral content (BMC) in younger motion segments (mean ± SD, 30.7 ± 11.1 g per motion segment *vs.* 27.8 ± 9.4 g). Increasing bone mineral content had a protective influence with each additional gram increasing survival times by approximately 12%.

Conclusion. Younger motion segments survive considerably longer when exposed to similar spine loading conditions that simulate repetitive lifting in neutral and flexed torso postures, primarily associated with the increased bone mineral content possessed by younger motion segments. Cycles to failure of young specimens at 22.5° flexion were similar to that of older specimens at 0° flexion, and survivorship of young specimens at 45° flexion was similar to the older cohort at 22.5°.

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Low back disorders (LBDs) are a major cause of both short- and long-term occupational disability in the United States,[1] and it is clear that workers in certain occupations are more susceptible to LBD.[2–5] Specifically, epidemiologic studies have shown that jobs involving heavy physical demands such as construction,[2,3] mining,[2,4] and farming,[5] engender increased risk of back pain. These occupations are thought to experience high LBD rates as a result of their extreme postural and manual lifting demands. Studies have shown that jobs involving significant lifting[6–9] and jobs that involve frequent bending[10–12] are associated with increased LBD risk.

Workers in the mining industry, in particular, often have to lift heavy materials in restricted workspaces that compel torso flexion. An understanding of the impact of repeated loading of the spine in flexion on fatigue failure of spinal tissues is thus a critical issue for our research agency, which is concerned with reducing the pain and disability associated with LBD in the mining industry.

It is well accepted that loads experienced by the spine during manual lifting tasks are sufficient to cause fractures in the endplates of lumbar vertebrae, particularly on repeated loading.[13–15] Endplate fractures may not be painful in and of themselves[16]; however, evidence suggests that the process of internal disc disruption and disc degeneration may be initiated *via* endplate fractures.[16,17] It has been shown that endplate damage will alter the distribution of stress in the disc, resulting in buckling of the lamellas of the internal anulus.[17] Once this process has been initiated, repeated spine loading will cause additional disruption of the anular fibers, resulting in formation of significant fissures or tears.[18–20] If these tears occur near or extend close to the outer portions of the anulus, a wound healing process is initiated, which involves infiltration of mast cells, macrophages, and release of cytokines and growth factors.[21] The presence of these infiltrates has been strongly linked to discogenic low back pain.[21,22] Indeed, for patients experiencing chronic low back pain, the prevalence of internal disc disruption is at least 39%,[19] which represents the most

common cause of chronic low back pain that has been demonstrated objectively.[16]

The ability to resist endplate fractures appears highly related to the amount of mineralized tissue of which the vertebra is comprised.[23–26] Studies have found that the relationship between the ultimate compressive strength of a vertebral body and bone mineral content (BMC) is essentially linear.[23,24,27,28] There is a decrease in vertebral strength with age, particularly beyond the age of 40 years, which is linked with a reduction in bone mineral in the vertebrae.[28,29] Specifically, there appears to be a significant loss of horizontally oriented trabeculae, particularly in the central portion of the vertebral body.[30,31] The departure of horizontal trabeculae deprives the vertically oriented trabecular "posts" of important reinforcement, and the load-bearing capability of the center of the vertebral body becomes degraded as a result.[32] The body appears unable to reconstruct the horizontal trabeculae; instead, bone is deposited in a manner to thicken or reinforce the vertically oriented members.[16] The decreased strength of the cancellous core of the vertebral body as aging takes place is associated with a shift in the load-bearing responsibilities from cancellous to cortical bone.[32] However, complex regional load-sharing responsibilities also appear to exist. Specifically, Eswaran *et al*[33] report that the cortical shell provides 45% of the support in the narrowest region of the vertebral body, but only 15% near the endplates.

The age-related changes detailed above undoubtedly impact the vulnerability of the endplates to fatigue failure. Specifically, the weakened trabecular network underlying the endplates permits increased deformation of the cartilaginous endplates and may increase the propensity to fracture.[32] The finding of increased fracturing in the endplates with increasing age appears to bear testimony to this development.[34] The purpose of the present investigation is to compare and contrast the fatigue failure responses of older motion segments to loads simulating a lift of 9 kg in 3 torso flexion postures (0°, 22.5°, and 45°),[15] to those of a younger cohort, and further, to evaluate the influence of BMC on fatigue life.

■ Materials and Methods

The biomaterials testing methods used for the younger cohort in this comparative analysis follow exactly those described in the article involving tests on the older spines,[15] where additional detail may be obtained. The current manuscript summarizes these procedures and additionally provides data on collection and analysis of BMC and bone mineral density (BMD), which were not detailed in the previous paper.

Three replications of a randomized block partially confounded factorial design were performed using a total of 18 lumbar spines (54 motion segments), none of which died of causes relating to spine pathology. The first 2 replications (12 spines or 36 motion segments) consisted of an older cohort of specimens (mean ± SD age, 80.7 ± 7.8 years).[15] This older sample was comprised of 8 male and 4 female spines. The third replication (6 spines or 18 motion segments) was performed using a younger group of specimens (mean ± SD age, 49.2 ± 17.3 years) and was comprised of 3 male and 3 female spines. Each spine was frozen in plastic bags until needed and subsequently defrosted before dissection into 3 motion segments (L1–L2, L3–L4, and L5–S1). Excess musculature, adipose tissue, and fascia were removed from the motion segments and ligaments spanning multiple levels were sectioned. Ligaments confined to a single motion segment were preserved.

Bone mineral density (BMD) and BMC for vertebral bodies L1–L4 of each spine were measured by means of dual radiograph absorptiometry using a Lunar DPX machine (Lunar, Madison, WI). Specimens were positioned on a bed of rice (to simulate body tissue) with the anterior aspect of the spine facing up. The BMD of the specimen was expressed in grams divided by the projected area of the bone. BMC was derived from the BMD and area measurements. BMD, projected area of the vertebral bone, and BMC for L1–L2 and L3–L4 segments individually were provided in standard reports. Table 1 provides information regarding the age of each specimen along with data on BMC and BMD for L1–L2 and L3–L4 motion segments from each spine.

Table 1. Age, Bone Mineral Content, and Bone Mineral Density for L1–L2 and L3–L4 Motion Segments From Old and Middle-Aged Lumbar Spines

Older Spines						Middle-Aged Spines					
		L1–L2		L3–L4				L1–L2		L3–L4	
Spine ID	Age (yr)	BMC (g)	BMD (g/cm^2)	BMC (g)	BMD (g/cm^2)	Spine ID	Age (yr)	BMC (g)	BMD (g/cm^2)	BMC (g)	BMD (g/cm^2)
M11	85	28.6	1.030	38.6	1.263	F20	55	13.9	0.615	18.8	0.679
M8	74	33.7	1.121	40.0	1.095	M23	60	39.6	1.152	53.7	1.264
M1	77	28.9	0.897	33.3	0.958	M21	61	25.0	0.793	29.5	0.793
F9	85	18.3	0.686	28.2	0.917	F22	42	33.5	1.368	40.0	1.317
M7	65	11.5	0.635	14.0	0.647	M18	60	29.4	0.991	39.0	1.140
F6	82	23.2	0.995	29.9	1.071	F19	17	20.5	0.932	25.8	0.999
F17	93	11.9	0.652	15.9	0.745						
M10	80	34.6	0.890	45.6	1.068						
F13	91	17.2	0.751	24.4	0.902						
M16	79	26.2	0.946	32.8	1.044						
M12	84	31.2	0.870	41.7	1.030						
M14	73	25.0	0.894	31.3	0.958						
Mean	80.7	24.2	0.864	31.3	0.975		49.2	27.0	0.975	34.5	1.032
SD	7.8	7.9	0.154	9.7	0.163		17.3	9.2	0.265	12.4	0.257

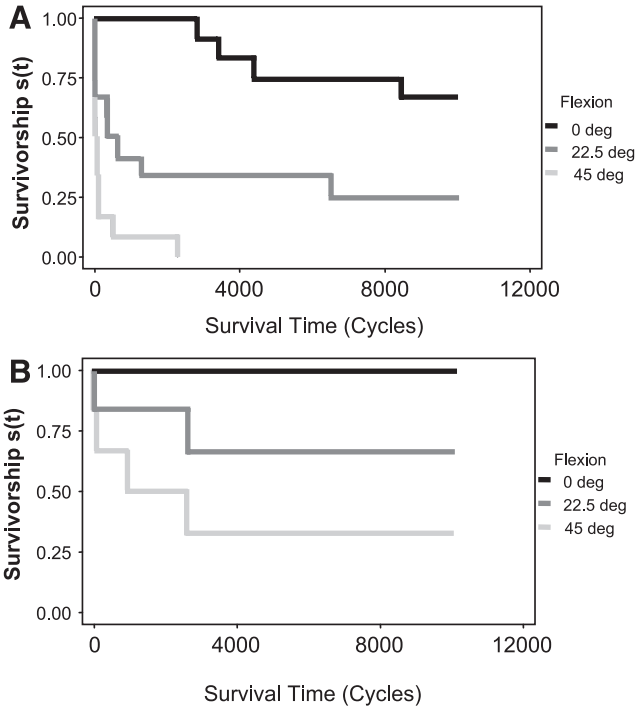

Figure 1. **A**, Survivorship functions for old spines by torso flexion angle (n = 36). **B**, Survivorship functions for young spines by torso flexion angle (n = 18). For each motion segment tested at 0° flexion, maximum load was 1300 N (load rate, 700 N/s), at 22.5° flexion maximum load was 2400 N (load rate, 2100 N/s), and at 45° flexion maximum load was 3150 N (load rate, 4800 N/s).

Anterior-posterior and left lateral radiographs of each motion segment were taken at 50 kV, 5 mA, and a film focus distance of 38 in. These initial radiographs served to detect the presence of existing defects in individual motion segments and were used to determine the relative angles of all 4 endplates of the motion segment. A dynamic EMG-assisted biomechanical model[35] was used to develop appropriate loads and load rates associated with lifting a 9-kg load starting at 3 trunk flexion angles: 0° (neutral), 22.5° (partial flexion), and 45° (full flexion). These spinal load estimates were obtained from an existing large database of lifting tasks. Resultant loads were 1300 N, 2400 N, and 3150 N for these torso flexion angles, respectively. Since the spine is composed of viscoelastic tissues, it was considered vital that the rate of loading also be correctly modeled in each posture. Average model estimates of load rate for the 3 postures were 700 N/s, 2100 N/s, and 4800 N/s, with increasing load rates in more flexed postures. Motion segments were flexed according to endplate angles obtained in a radiographic study of subjects performing torso flexion *in vivo*.[36]

Motion segments were potted in trays containing polymethylmethacrylate, the flexion angle of each motion segment being confirmed by endplate measurements obtained *via* multiple radiographs during the fixation process. This flexion angle was maintained constantly throughout the cyclic loading regimen for a given specimen. All tests were conducted in a humidified environmental chamber at a temperature of 37 C. Motion segments were statically loaded for 15 minutes (at 500, 750, or 1050 N depending on torso flexion angle) and then cyclically loaded at 0.33 Hz until failure or the maximum number of cycles (10,020) was completed using a hydraulic materials testing machine (MTS Systems Corp., Eden Prairie, MN). Failure was defined as a displacement of 10 mm after termination of the period of creep deformation. The primary dependent measure was the number of cycles to failure.

Comparisons of the fatigue failure responses of specimens were achieved *via* survival analysis techniques, specifically *via* Kaplan-Meier Product Limit Estimation and Cox Regression Analysis. Kaplan-Meier Product Limit Estimation, and mean survival times were obtained separately on the younger specimens (18 motion segments) and older specimens (36 motion segments). Plots of survivorship functions for each group were obtained. Variables included in the Cox regression models included simulated torso flexion angle, lumbar level, BMC, BMD, and weight of the motion segment. Both young and old specimens were included in Cox models to better evaluate the influence of a range of bone mineral values. Since this regression analysis incorporated bone mineral data, L5–S1 segments (from which BMC data were not obtained) were omitted from this analyses. Alpha levels for all tests of significance were set at 0.05.

■ Results

Kaplan-Meier Product-Limit estimation demonstrated longer survival times and an increased number of censored observations (*i.e.*, the number of motion segments surviving the entire 10,020 cycles of the test) for the younger cohort of specimens compared with the older group. Figures 1A and B illustrate the survivorship functions *s(t)* at each angle of torso flexion for older and younger spines, respectively. As can be seen from these figures, increased loads and load rates associated with greater torso flexion were associated with decreased survival of specimens in both groups. However, the survivorship functions for younger spines show a dramatically improved fatigue life for the simulated lifting task at all torso flexion levels. Table 2 details the difference between these groups in terms of censored observations and mean survival time. In all cases, survival was significantly enhanced in the younger sample.

Table 2. Comparison of Censored Observations and Mean Survival Times for Old and Middle-Aged Spine Motion Segments Using Spinal Loads Predicted When Lifting a 9-kg Load in Three Torso Flexion Postures

	0°		22.5°		45°	
	% of Censored Observations	Mean Cycles to Failure	% of Censored Observations	Mean Cycles to Failure	% of Censored Observations	Mean Cycles to Failure
Older specimens (n = 36)	67	8267	25	3261	0	263
Middle-aged specimens (n = 18)	100	10,020	67	7124	33	3929

Table 3. Results of Cox Regression Models Incorporating Effects of Simulated Torso Lifting Loads at Different Angles of Flexion and Bone Mineral Content of the Specimens

	df	Beta	SE	*P*	Risk Ratio	95% CI for RR
Flexion	2			0.001		
22.5°	1	2.756	0.862	0.001	15.741	2.905–85.309
45°	1	3.923	0.921	0.000	50.530	8.310–307.245
BMC	1	−0.127	0.036	0.001	0.881	0.820–0.946

Table 3 provides the results of Cox regression models examining the effects of flexion angle and BMC. No other variables input to the model were significant predictors of time to failure. On the average, younger spines were found to possess approximately 3 additional grams of bone mineral per motion segment than older spines (mean ± SD, 30.7 ± 11.1 g *vs.* 27.8 ± 9.4 g).

As can be seen in the Cox regression analysis presented in Table 3, both flexion angle and BMC were significant predictors of the time to failure. Compared with the referent posture (0° flexion), simulated lifting in 22.5° flexion had a 15.7-fold increase in relative risk, while the 45° condition saw a 50.5-fold increase in relative risk compared with the referent condition. As can be seen from the negative exponent, increasing BMC exerted a protective influence. According to the model, every gram of BMC resulted in a 12% increase in cycles to failure for this sample (when controlling for the effects of flexion).

Table 4 presents a comparison of failure mechanisms observed in specimens failing *via* fatigue in the older *versus* the younger age samples. Of those specimens failing *via* fatigue, the proportion of younger specimens experiencing endplate failure appeared somewhat lower in the more erect postures (0° and 22.5° flexion); however, a similar proportion of endplate failures were observed at 45° flexion in both groups. Frank disruption of the zygapophysial joints was observed in 7 of the 25 older motion segments failing *via* fatigue but was never observed in the younger specimens. Increased laxity of the zygapophysial joints was observed in 3 of the younger specimens failing *via* fatigue, however. The sole disc failure observed in fatigue failure specimens was observed in one of the younger motion segments exposed to the 22.5° torso loading condition. In general, older specimens tended to exhibit more extensive damage (*i.e.*, multiple failure modes) as compared with younger specimens.

■ Discussion

Failure of spinal tissues may be considered a function of load magnitude, loading frequency, and load tolerance (or strength) of the tissue. The current investigation provides data to address all 3 of these potential factors in an *in vitro* experiment and provides some insight to the changes in fatigue tolerance that occur with aging. Results of this study indicated that younger (middle-aged) spines were found to have considerably increased fatigue life at all 3 flexion angles compared with an elderly cohort. However, within each age cohort, the increased spinal load associated with lifting in deeper torso flexion resulted in a remarkable reduction in cycles to failure. None of the middle-aged specimens failed at the 0° torso flexion simulated lift. It may be observed that, for most materials, repetitive loading at less than 30% of the material's ultimate strength will not typically result in fatigue failure.[37] This is sometimes referred to as the "endurance limit" associated with cyclic loading. One might infer that the 1300 N load at the 0° flexion condition was below the endurance limit of all of the younger specimens tested. Furthermore, while none of the older sample survived the maximum of 10,020 cycles at the 45° torso flexion simulated lift, one third of younger specimens were able to do so. Comparison of the survivorship functions reveals that survival of young specimens at 22.5° flexion was quite similar to that of older specimens at 0° flexion, and that the survivorship of young specimens at 45° was similar to that of the older cohort at 22.5° flexion. It would appear that the diminished BMC in the older specimens was largely responsible for differences in survival between the 2 groups.

Unfortunately, the open-celled sponge-like structural design of vertebral bone, while being able to deform

Table 4. Comparison of Fatigue Failure Mechanisms of Older *Versus* Middle-Aged Motion Segments by Flexion Angle

	0°		22.5°		45°	
	Older	Middle-Aged	Older	Middle-Aged	Older	Middle-Aged
No. of specimens tested	12	6	12	6	12	6
No. of specimens failing *via* fatigue	4	0	9	2	12	4
Specimens exhibiting endplate failure						
Inferior EP (superior vertebra)	2	0	9	1	10	3
Superior EP (inferior vertebra)	0	0	6	0	6	2
Specimens exhibiting zygapophysial joint damage						
Increased laxity	0	0	3	1	4	2
Disruption	2	0	3	0	2	0
Specimens exhibiting vertebral body fracture						
Superior VB	2	0	5	1	6	4
Inferior VB	0	0	1	1	2	0
Specimens exhibiting disc failure	0	0	0	1	0	0

more without cracking (compared with long bones), is not as adept at withstanding high loads.[38] When spinal loads are sufficiently large (as in lifting tasks), microfractures in the trabeculae will occur, invoking the process of bone remodeling. In trabecular bone, remodeling results in substantial bone loss (especially compared with cortical bone) due to higher number of remodeling sites per unit volume, along with the fact that new bone formation lags behind the ability to resorb bone starting early in midlife. This process will lead to a loss of trabecular plates and connectivity, resulting in substantial strength loss.[39] More rapid remodeling is associated with decreased bone quality and increased risk of fracture, due to the decreased density of newly remodeled bone tissue, more unfilled sites where bone has been resorbed, along with poorer cross-linking of collagen fibers.[38,40] The decreased fatigue life exhibited by older motion segments is an unfortunate consequence of the depletion of bone mineral resulting from this slow but steady process.[23,30] While the absolute difference in BMC was small between young and old specimens (less than 3 g per motion segment, on average), the architectural and structural importance of even small changes in BMC was dramatically apparent in this study.

It is interesting to note that this study found BMC to be a better predictor of vertebral integrity than BMD, a finding also observed elsewhere.[41] One reason this may be true is that BMC provides a combined measure of both the size and density of vertebral bone, whereas BMD obscures the influence of the specimen size, known to be positively correlated with vertebral strength.[38,40] BMC may thus be a superior measure to use when assessing or predicting vertebral strength. It should not escape notice that BMC is easily obtained *in vivo*, and the findings of this study could have potential clinical relevance in terms of assessing (at least in general terms) a patient's risk of damage to spinal tissues, especially if the patient is engaged in physically demanding lifting tasks. For example, a job requiring repetitive lifting in flexed postures in a person with low BMC would clearly be a recipe for rapid failure of spinal tissues.

It is incumbent on us to consider the relevance of the current results with respect to potential low back pain pathways. Indeed, results of this study may have relevance to different pain mechanisms, depending on the physiologic age of the spine. One possible pathway to pain is that fatigue failure will lead to endplate fractures leading to disc degradation, as detailed in the introduction. If one considers a healthy young spine (with no disc degeneration and with a normal "young" BMC value), results of this study suggest relatively little risk would be associated with lifting a 9-kg load repeatedly as long as an upright posture could be adopted. However, risk of endplate failure in such young spines would increase substantially when flexed lifting is performed, although a relatively high number of cycles may be necessary when lifting a moderate load. Lifting of heavier loads would obviously accelerate this process, and it should be noted that, with a sufficiently heavy load, one would not anticipate the upright posture to remain consistently protective. In young spines, trabecular damage (particularly in the vertebral centrum[42]) may lead to decreased endplate support leading to cracks of the endplate, mortared *via* scar tissue, and initiating a degenerative cascade in the disc. It should be noted that the majority of studies examining the association between heavy physical loading and disc degeneration have found an association,[43–55] although it should be noted that this association has not been universally observed.[56–58]

The role of inherited traits on disc degeneration has received increased attention in recent studies[44,59] and merits some discussion with respect to the current research findings. Investigators in these studies examined monozygotic (MZ) twins discordant for physical loading, and evaluated measures of disc degeneration *via* magnetic resonance imaging. Although physical load was found to have a significant effect on disc degeneration in these investigations, it was asserted that factors related to inheritance accounted for a more sizeable portion of the variability in disc degeneration. In the context of the present experiment, one inherited trait that may help explain this result is that of BMC. It would seem intuitive that MZ twins, sharing an identical DNA blueprint, would also share a high correspondence in BMC (*i.e.*, would have comparable vertebral size and bone density). Indeed, studies over the last 30 years in healthy twins consistently demonstrate a large genetic contribution to bone mass.[60–65] Heritability estimates of BMD in the lumbar spine have been reported at 89%, and lumbar anthropometric dimension heritability estimates range from 61% to 83%.[65] Thus, it would seem quite plausible that a high proportion of the variability in disc degeneration attributed to inheritance in these studies may, indeed, be due to differences in vertebral strength between twin pairs. It would be a reasonable supposition, for example, that twins with higher BMC would possess endplates that would be less prone to fracture (due to enhanced structural support) and less likely to lead to disc degeneration as a result. Conversely, twins possessing low BMC would be expected to experience more rapid endplate failure and disc degeneration. As demonstrated in the current study, small differences in BMC can have an extraordinary impact on vertebral strength and fatigue life. Thus, inheritance of bone mineral attributes may play a large role with respect to the ability to tolerate physical loads on the spine, which may impact the development of disc degeneration.

The likelihood that a high correlation in BMC exists within MZ twin pairs raises a related issue regarding the impact of an identical twins design in evaluating the influence of physical loading on disc degeneration. It should be readily apparent that tissue failure will be influenced both by the magnitude of an imposed load and strength of the material on which the load is exposed. One would anticipate that the effect of this association would be most prominent when both factors are free to

vary, and particularly when both variables are at their discordant extremes (*i.e.*, low-load/high material strength may produce little or no damage, whereas high-load/low material strength may produce significant damage). Thus, the effect of changes in magnitudes of physical loading on tissue damage would be expected to be more apparent when examined in a context of material strengths that comprise a wide range of values. However, it should be recognized that in a twin design, because of a high induced correlation in BMC (*i.e.*, material strength) between twins, the impact of changes in physical load are examined in a somewhat restricted context (only within materials of similar strength). While such comparisons are certainly legitimate, they may not be fully representative of the effect that changes in physical load would bring to bear if examined over a wide range of material strengths. This may help to explain why physical load was found to have a lesser influence on degenerative changes in the twin studies[44,59] (where physical load effect was restricted to matched levels of material strength) compared with other epidemiologic studies (where differences in physical loading were examined without restrictions in the range of material strength).[43–55]

Disc degeneration will alter the mechanical properties and load distribution within the spinal column, and as this process evolves with age, additional threats may emerge that may lead to low back pain when exposed to repetitive loading. Regional bone loss appears to be related to the degree of disc degeneration, with greater bone loss seen in the centrum of the vertebra with increased disorganization of the disc. This may be due to a redistribution of load to the vertebral body periphery resulting from disc degeneration[30] and the development of stress concentrations in the disc.[66] The resulting weaknesses in the vertebrae may lead to osteoporotic fractures, which may be a particular risk when the older, osteoporotic spine is loaded in a flexed posture.[67] These fractures may result in significant pain, deformity, and disability.[68]

It is important to note that the number of cycles until the failure criterion was reached in this biomaterials testing protocol should not be taken as representing a value that may be sustained before clinically relevant damage occurring. Indeed, endplate fractures may be initiated with less compaction than allowed in the present protocol and will usually occur with less that 5 mm.[16,17] We established a higher displacement criterion in our initial experiment due to the older sample and a desire to maximize experimental variability in fatigue life to better establish the influence of torso flexion. It is not precisely known how mechanical tests of cadaver specimens relate to *in vivo* loading. Some have suggested that the healing capabilities of bone *in vivo* may result in less rapid failure than during *in vitro* testing.[17,69] Others have suggested that osteoclastic resorption in the physiologic state may actually reduce strength and accelerate *in vivo* fatigue failure compared with that observed during *in vitro* testing.[26,70]

While the relationship of *in vitro* testing to the physiologic state is not entirely clear, results of the present investigation provide several important findings that may have clinical relevance with respect to cyclic loading and failure of spinal tissues. For example, results of this study suggest that exposure to spinal loads experienced in flexed lifting of a given weight are likely to result in much more rapid fatigue failure compared with the reduced loads experienced in upright lifting. These findings provide strong support for minimizing the amount of forward bending performed in lifting tasks, especially in heavy industry such as mining. Future lifting guidelines should be designed to account for the rapid fatigue failure that may occur in flexion. Results also suggest that older workers will experience fatigue failure much more quickly, primarily due to the reduced BMC in their spines. This suggests that future lifting limits may also need to consider age-related changes to prevent tissue damage as bone loss continues to accumulate.

The results presented in this study are not encouraging for mine workers who are bound to adopt a flexed postures during lifting tasks due to restrictions in vertical space often present in the underground mining environment (particularly in coal mines with working heights at or around 1.5 meters). However, these findings may help explain the high incidence rates for LBD typically observed in mining.[2,4] Manual materials handling is a pervasive activity in mining and if workers are forced to perform such activities in a flexed posture, the results here suggest more rapid failure of spinal tissues, potentially accelerating the process of disc degeneration. There are unfortunately limited postural alternatives in restricted spaces and it is more difficult (though not impossible) to provide mechanical-assist devices in restricted spaces. Lifting in a kneeling posture will help keep the spine in a more upright orientation and may be a useful alternative to stoop lifting; however, lifting capacity is reduced in the kneeling posture and there may certainly be a trade-off with regard to increased risk of knee disorders. Clearly, results of this study suggest that loads handled in a stooped posture be minimized to the greatest extent possible. Furthermore, the need for new and better mechanical aids that can be effectively utilized in restricted workspaces would seem to be a critical element in reducing LBD risk in underground mines.

Certain limitations should be considered regarding the data presented here. These include the possibility that preexisting medical conditions or loading histories might have differentially affected the specimens. The chances of prior damage would certainly be greater in older specimens. In this regard, it is important to realize that, although fatigue failure occurred more rapidly in older specimens, this does not necessarily imply greater back pain experience in older osteopenic spines. It is quite possible (in fact likely) that such spines have already

experienced the degenerative changes that may have led to pain much earlier in life.

■ Key Points

- The fatigue failure responses of 18 working age motion segments (average ± SD age, 49 ± 17 years) exposed to spines loads simulating lifting tasks at 3 trunk flexion angles were compared with those of a previously tested elderly cohort (average ± SD age, 81 ± 8 years).
- Compared with the older sample of spines, the younger sample exhibited a substantial increase in cycles to fatigue failure in all the torso flexion conditions.
- Bone mineral content was greater in the younger motion segments, 30.7 g (±11.1 SD) per motion segment, *versus* 27.8 g (±9.4 SD) in older spines.
- Cox regression modeling indicated increasing relative risk of fatigue failure for segments exposed to simulated spinal loads when lifting at increased angles of torso flexion (risk ratio for 22.5° flexion = 15.7 compared with 0°; risk ratio for 45° = 50.5 compared with 0°).
- Increased bone mineral content was found to have a protective influence (*i.e.*, every increase of 1 g BMC led to 12% increase in cycles to failure).

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