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Factors Associated With Kyphosis Progression in Older Women: 15 years experience in the Study of Osteoporotic Fractures

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Abstract

Age-related hyperkyphosis is thought to be a result of underlying vertebral fractures, but studies suggest that among the most hyperkyphotic women, only one in three have underlying radiographic vertebral fractures. Although commonly observed, there is no widely accepted definition of hyperkyphosis in older persons, and other than vertebral fracture, no major causes have been identified. To identify important correlates of kyphosis and risk factors for its progression over time, we conducted a 15 year retrospective cohort study of 1,196 women, aged 65 years and older at baseline (1986–88), from four communities across the United States: Baltimore County, MD; Minneapolis, MN, Portland, Oregon, and the Monongahela Valley, PA. Cobb angle kyphosis was measured from radiographs obtained at baseline and an average of 3.7 and 15 years later. Repeated measures, mixed effects analyses were performed. At baseline, the mean kyphosis angle was 44.7 degrees (standard error 0.4, standard deviation 11.9) and significant correlates included a family history of hyperkyphosis, prevalent vertebral fracture, low bone mineral density, greater body weight, degenerative disc disease, and smoking. Over an average of 15 years, the mean increase in kyphosis was 7.1 degrees (standard error 0.25). Independent determinants of greater kyphosis progression were prevalent and incident vertebral fractures, low bone mineral density and concurrent bone density loss, low body weight, and concurrent weight loss. Thus, age-related kyphosis progression may be best prevented by slowing bone density loss and avoiding weight loss.

Keywords

kyphosis; hyperkyphosis; kyphotic posture; hunchback; causes

Introduction

Hyperkyphosis, or an increased thoracic curvature, is commonly observed in older persons affecting up to 40% of older women, depending on the cut-off criterion used to define accentuated curvature (1). While the development of age-related hyperkyphosis is often attributed to underlying spinal osteoporosis, only 36–38% of those with the most abnormal kyphosis have underlying fractures (2, 3). Remarkably little is known about its epidemiology, including a lack of a standardized definition as to what degree of kyphosis should be defined as hyperkyphosis. Furthermore, risk factors for kyphosis progression in the general population are unknown, as most studies to date have been small in scale, cross-sectional in design, and/or primarily focused on women with osteoporosis (1). Other than vertebral fractures, degenerative disc disease (3–5) and muscle weakness have been reported to be associated with worse kyphosis (6–7), but none of these studies have been prospective in design.

While the causes of age-related kyphosis progression are not well understood, there is growing evidence that women with increased kyphosis experience poorer health and earlier death (1, 8). Regardless if kyphosis is measured qualitatively or quantitatively from radiographs, compared with unaffected women, women with hyperkyphosis have poorer pulmonary and physical function, and are at higher risk of sustaining non-spine fractures (9–13). Given these associations between hyperkyphosis and ill-health, a number of recently published small trials have investigated therapeutic options to prevent or reverse kyphosis progression (14–17).

Missing from our current understanding of age-associated hyperkyphosis is basic knowledge of its natural history and the identification of risk factors for its progression over time. Therefore, using data from the Study of Osteoporotic Fractures (SOF), we sought to: 1) identify important correlates and describe the natural progression of thoracic kyphosis; and 2) identify important, and potentially modifiable causes of kyphosis progression in 1,196 older women followed for an average of 15 years.

Methods

Participants

Data from the Study of Osteoporotic Fractures (18) were used to examine hypothesized factors associated with kyphosis and kyphosis progression in older women. For the current study, we randomly selected 1,000 participants from 9,334 cohort members who had baseline and follow-up radiographs of the thoracic spine that were done an average of 3.7 and 15 years later. Originating in September 1986, SOF was the first and largest prospective U.S. study devoted primarily to the study of fractures in 9,704 older primarily Caucasian women aged 65 years and older recruited from 4 areas of the United States (Baltimore County, Maryland; Minneapolis, Minnesota; Portland, OR; and Monongahela Valley, Pennsylvania). SOF women were recruited irrespective of bone mineral density (BMD) and fracture history; those unable to walk without assistance and with bilateral hip replacements were excluded from study.

To address issues of survivor bias, an additional random sample of 200 women who did not have all three films were also included. The final sample size was 1,196 after excluding 4 women without complete Cobb angle data.

Measurements (see Table 1)

Vertebral Radiographs and Morphometry—At the baseline visit and follow-up visits an average of 3.7 years and 15 years later, radiographs of the thoracic and lumbar spine were

obtained in accordance with the 1995 National Osteoporosis Foundation guidelines (19). Recumbent lateral thoracic spine plain films were taken with a tube-to-film distance of 40 inches, centered at T8 for thoracic films and at L3 for lumbar films.

Cobb Angle Kyphosis—We used a modified Cobb angle method to measure kyphosis from recumbent lateral thoracic spine plain films. Using a translucent digitizer (GTCO, Rockville, MD) and cursor, a technician marked four points corresponding to the four corners of the vertebral body at T4 and T12, and the X, Y coordinates for each point were recorded on an electronic grid with a resolution of 0.1 mm. While the original Cobb method uses angles of inflection to determine the superior and inferior margins for line placement (20), the modified Cobb method uses a fixed cut-off of T4 and T12, largely because T1 to T3 are usually not well visualized on lateral spine films. From the superior surface of T4 and inferior surface of T12, a computerized digitization program erected perpendicular lines, the intersection of which is the kyphotic angle. The intra-rater reliability ICC for repeated digitized Cobb angle readings was 0.984 (21).

Prevalent Vertebral Fracture—Trained technicians triaged radiographs as normal, uncertain, or probably fractured on the basis of a semiquantitative grading scheme (22). For those classified as uncertain, the study radiologist then determined whether these films had normal or probably fractured vertebra(e). Films categorized as probably fractured were evaluated by six point vertebral morphometry, the methods for which have been described previously in detail (23).

Incident Vertebral Fracture Assessment—An incident fracture was diagnosed if any of 3 vertebral heights (anterior, middle, or posterior) decreased by more than 20% and by at least 4mm compared with the previous film.

Vertebral Wedging Index—In addition to the prevalent and incident vertebral fracture assessments, the anterior to posterior vertebral height ratio was calculated for each vertebral body from T4 to L4, and averaged over the 13 vertebrae to create the participant's vertebral wedging index, a measure of overall vertebral wedging deformity in the individual. Change in the individual's vertebral wedging index from baseline was computed at both follow up visits.

Disc Height Ratio—The thoracic spine radiographs used to measure the Cobb angle, were also used to calculate intervertebral disc heights between T4 and T12. Two heights were calculated for each disc space: 1) anterior height (H_a) and 2) posterior height (H_p). A total of 8 disc spaces were measured, with two disc height ratios at each interspace calculated to define two types of deformity: 1) the disc wedging ratio, H_a/H_p and 2) the disc height compression ratio, $(2H_{a,i} + 2H_{p,i})/(H_{a,i-1} + H_{p,i-1} + H_{a,i+1} + H_{p,i+1})$. For the uppermost and lowermost intervertebral levels, we used only one adjacent disc level for comparison. For use in supplementary analyses, the average disc wedging ratio over all 8 disc spaces was computed for each participant at each visit, and change in the average ratio from baseline was calculated at both follow up visits.

Degenerative Disc Disease—A disc was defined as having degenerative disease if the disc wedging ratio was greater than 3 standard deviations below the level-specific sample mean or the disc height compression ratio was less than 0.80, and there was no fracture in the vertebral bodies on either side of the disc. This last requirement was included to avoid misreading compensatory disc changes as degenerative disease. An individual was classified as having degenerative disc disease (DDD) at the baseline visit if one or more of the 8 discs met the above degenerative disc criteria.

Other Measurements—All study participants answered questions contained in baseline comprehensive and follow-up questionnaires administered at the 3rd and 8th clinic visits. Basic demographic information including age, clinic site, and health behaviors such smoking (never versus past/present), alcohol (drinks per week, adjusted for atypical drinking), and physical activity (estimated total kcalories burned per week in the past year) habits were ascertained. A family history of hyperkyphosis in each parent was assessed by the question, "Did your natural mother (or father) develop a `dowager's hump' or a spine that was stooped or bent forward?"

At the baseline visit, participants had their height measured by the Harpenden stadiometer, gait speed timed over 6 meters, and weight assessed by balance beam scale in light clothing. Upper body isometric grip strength in both the dominant and non-dominant hand was measured using a hand-held isometric dynamometer (Sparks Instruments and Academics, Coralville, Iowa). Follow-up measures of timed gait speed, weight, and grip strength were repeated an average of 3.7 and 15 years later. At the baseline visit and fourth follow-up visit an average of 5.7 years later, calcaneal bone mineral density was measured by single photon absorptiometry (OsteoAnalyzer; Siemens-Osteon, Wahiawa, Hawaii). We used the cBMD measurement instead of spine BMD because only the former was contemporaneous with the baseline kyphosis assessment (in SOF, the initial spine BMD measurement was not done until the second clinic visit that occurred 2 to 4 years after the baseline kyphosis measurement).

Statistical Analysis

We used linear, mixed effects regression to model the progression of kyphosis over time as an individual-specific growth curve with three parameters: starting value (baseline or intercept), short-term change (between baseline and 3.7 years), and long-term change (between baseline and 15 years). We used a 3-parameter growth curve because the mean annualized rate of progression of kyphosis in the cohort was faster between baseline and first follow-up, than between first and second follow-up, and because we hypothesized that baseline values of time-varying predictors (such as body weight, bone mineral density, and vertebral fractures) would have stronger associations with short-term than long-term progression of kyphosis.

Characteristics of the study participants included in these analyses were compared to the rest of the participants in the parent study cohort using T-tests and Chi-squared tests for continuous and categorical variables, respectively (Table 2). To account for the potential problem of survivor bias (in estimates of baseline kyphosis and short-term progression), the 146 participants in our sample who did not survive through year 15 and the 1,050 who did were differentially weighted to match the complete SOF cohort's probability of survival through year 15. After being normalized (to sum to the sample size, 1196), survivors got a weight of 0.66 and non-survivors a weight of 3.41.

Because we hoped to identify modifiable factors for kyphosis progression, in addition to known covariates such as age, vertebral fracture, DDD, and muscle strength, we incorporated hypothesized, but previously untested variables into our model. All three growth curve parameters were modeled to vary by baseline values of: 1) age; 2) clinic site; 3) prevalent vertebral fracture (yes/no and number of prevalent vertebral fractures); 4) calcaneal BMD (cBMD); 5) degenerative disc disease (DDD); 6) family history of hyperkyphosis; 7) grip strength; 8) gait speed; 9) smoking; 10) alcohol use; 11) physical activity level; and to vary from woman to woman as random effects. In addition, we allowed for within-person changes (during the study) in cBMD, grip strength, gait speed, body weight, and incident morphometric vertebral fractures to affect contemporaneous changes in kyphosis. The random effect variances in the three growth curve parameters (intercept or

baseline kyphosis, short-term change in kyphosis, and long-term change in kyphosis) were each examined before and after inclusion of covariates/predictors to determine the proportion of variance explained by the complete set of predictors. The proportion of variance explained was calculated for each parameter as: (variance in null model - variance in full model) / (variance in null model).

Since it has been postulated that hyperkyphosis is caused primarily by deformities in vertebral bodies and inter-vertebral discs (24), in supplementary analyses, we added vertebral wedging index (continuous) at baseline, change in vertebral wedging index from baseline, average disc wedging ratio at baseline (continuous), and change in average disc wedging ratio from baseline to the full model, and examined the improvement in % variance (of baseline kyphosis, short-term change, and long-term change) explained. SAS software was used for all analyses (SAS Institute, version 9.1, Cary, NC).

Results

Women were an average age of 69.1 (standard deviation [SD] = 3.7) years at the baseline visit and had a mean kyphosis angle of 44.7 (standard error in the mean [SE] = 0.4; SD =11.9) degrees. Most women were either of Northern, Central or Southern European descent, with European ethnicities comprising more than 95 percent of the study cohort. Study participants were on average, younger than the parent cohort, were slightly taller with better BMD and had fewer prevalent vertebral fractures (Table 2). They also were more likely to report better overall health and performed better on physical function measures of grip strength and walking speed.

Kyphosis progressed an average of 2.6 (SE = 0.2; SD = 4.0) degrees between baseline and the first re-assessment an average of 3.7 years later, and progressed an average of 7.1 (SE = 0.3; SD =6.8) degrees between baseline and second re-assessment, an average of 15 years later (Figure 1- see main trajectory).

Bivariate associations between characteristics and kyphosis and kyphosis progression are reported in Table 3. Notable findings include that with each prevalent vertebral fracture, kyphosis is worse by an average of 4.3 degrees. A similar magnitude of 4.5 degrees increase in kyphosis is seen with incident vertebral fracture.

In the full multivariable model that included all major hypothesized predictors of kyphosis and kyphosis progression, prevalent vertebral fracture, DDD, family history of hyperkyphosis, and greater body weight were each associated with greater baseline kyphosis, while high cBMD, and current smoking were associated with less baseline kyphosis (Table 4). We determined that with each prevalent vertebral fracture, baseline kyphosis increased by 3.3 degrees (95% CI: 2.3 – 4.4). Greater body weight was associated with less short-term progression of kyphosis while higher baseline BMD predicted less long-term kyphosis progression. The only predictor of both worse short-term and long-term kyphosis progression was the presence of a prevalent vertebral fracture (Table 4 and Figure 1 – see curves of women with and without prevalent vertebral fractures).

In addition, new vertebral fractures after the baseline examination were associated with subsequent increase in kyphosis (average increase of 3.8 degrees), while increases in cBMD and body weight were associated with reduction in kyphosis. Other potential correlates such as gait speed, alcohol use, grip strength, grip strength change, and self-reported physical activity were not significantly associated with baseline kyphosis or kyphosis progression. Overall 12% of the variance in baseline kyphosis, 30.3% of the variance in short-term change, and 30.8% in long-term change in kyphosis was explained by the full model.

In supplementary multivariable analyses (not shown) that substituted the count (1,2,3, etc.) for the simple presence of prevalent vertebral fracture (yes/no), each fracture increased kyphosis by 3.3 degrees (95% CI: 2.3 – 4.4). Another supplementary analysis that included vertebralwedging index at baseline, change in vertebralwedging index from baseline, average disc wedging ratio at baseline, and change in average disc wedging ratio from baseline in the model increased the % variances explained to 89% for baseline kyphosis, 55.0% for short-term change, and 40.8% for long-term change.

Discussion

We found that over an average of 15 years of follow-up, kyphosis in older women increased by about 7 degrees. As expected, prevalent and incident vertebral fractures were significant contributing factors towards kyphosis progression. With each prevalent fracture, kyphosis increased 3.3 degrees and women who experienced one or more incident vertebral fractures had an increase of 3.8. Similar to some previous reports, we found that BMD and DDD were associated with baseline kyphosis (3, 25). Novel, newly identified predictors include a family history of hyperkyphosis, weight, and smoking which were associated with baseline kyphosis and low bone density, concurrent bone density loss, and concurrent weight loss which predicted kyphosis progression over time.

Compared to women without a family history of hyperkyphosis, women who reported a positive family history were more likely to have worse kyphosis. While the effect was only 2.6°, it remained a significant predictor in even the fully adjusted more inclusive multivariable model, and the magnitude of the effect was unchanged.

There has been some controversy as to whether BMD itself is associated with hyperkyphosis (6, 8–9, 26). Our study demonstrates that BMD is not only associated with kyphosis at baseline, but that it is a strong and significant predictor of long-term progression over 15 years. Per 0.1 gm/cm^2 decrement in BMD, women had a 2.3° greater baseline kyphosis and a 1.1° greater long-term increase in kyphotic angle. In addition to a low baseline value of BMD, contemporaneous change in BMD was also significantly associated with kyphosis progression over time (per 0.10 gm/cm^2 loss, there was a 2° kyphosis progression). These findings suggest that low BMD and continued bone density loss, independent of vertebral fractures, are important in determining kyphosis progression. The calcaneal BMD measurement largely reflects trabecular bone that is also mainly present in vertebral bodies; ongoing trabecular bone loss, likely contributes to vertebral wedging (that can range from a non-fracture deformity to a wedge compression fracture) that results in worsening kyphosis. To support this theory, adding a continuous measure of vertebral body height ratios along with disc height ratios to our models increased the variance explained in baseline kyphosis by 77% and in long term progression by 10%.

Few studies have investigated how degenerative disc disease may contribute to hyperkyphosis in older persons, but they all report a significant correlation between anterior disc height loss and kyphosis (3–5). These were all cross-sectional investigations, and it was unclear how DDD might affect kyphosis progression. In the current study, our findings confirmed that baseline DDD is a strong predictor of baseline kyphosis, as women with DDD had 3.5° more kyphosis than women without DDD. We also found that the effect of increased age on kyphosis severity was accounted for by DDD, a finding that has not been described previously. In multivariable models that included all covariates but DDD, age remained a significant predictor of worse baseline kyphosis; however, once DDD was added to the model, age loss significance. Unlike the baseline bone density measurement, however, the baseline measure of DDD did not predict kyphosis progression over time.

A new finding resulting from this study was the correlation between weight, weight loss and kyphosis and its progression. Interestingly, a greater baseline weight was significantly associated with greater baseline kyphosis, although the effect was small and by 3.7 years of follow-up, a greater baseline weight predicted less kyphosis progression. The positive association between baseline weight and baseline kyphosis might reflect the impact of increased weight on posture in older adults who typically have lower muscle mass for a given body weight than younger individuals. With regards to weight change, with each five kilograms of loss, there was a 1.0° increase in kyphosis. Weight loss in older women is associated with bone loss and fractures (27, 28), and it comprises one of the core components of a frailty index (29, 30). A picture of an elderly hyperkyphotic woman connotes an image of frailty, and weight loss may play a key role in the development of both. Older persons who lose weight, preferentially lose more muscle over fat (31); with loss of core muscle support, kyphosis would be expected to progress. Although we did not find that grip strength was associated with kyphosis, other evidence demonstrates that older men and women with poor spinal muscle composition suffer from hyperkyphosis (32). In our study, weight status is one newly identified potentially modifiable risk factor of hyperkyphosis development.

Both in the unadjusted and fully adjusted models, current smoking was associated with less kyphosis at baseline, but not with kyphosis progression. While smoking is usually associated with negative disease outcomes including chronic obstructive lung disease and cancer, there are a few examples of medical conditions where smoking may be protective, such as in preventing Parkinson's disease (33, 34) and ulcerative colitis (35) . With regards to kyphosis, in the Rancho Bernardo Study, there was a trend that smokers tended to have less kyphosis (36).

The overall clinical significance of our findings stem from being able to demonstrate that per vertebral fracture, kyphosis increases an average of 3 – 4 degrees. Having DDD increases kyphosis by an average of 3.5 degrees. Extrapolating from our previous work that demonstrated that per standard deviation increase in kyphosis, all-cause mortality rate (hazard) increased by 15% (95% CI: 1% – 30%), we estimate that women with 3 degrees (or 0.25 SD) more kyphosis would have 3.6% (95% CI: 0.25% –6>8%) higher mortality rate (8).

Our study has some limitations. First, because the study participants were older and selected because they had repeated radiographs done over a 15-year period, they were likely among the healthiest participants among an already high functioning study population and the study inclusion criteria could have introduced the problem of survivor bias. To minimize this potential problem, we selected a subsample of participants who didn't survive the entire period and performed weighted analyses. Second, it is now known that postural problems affect both sexes and the SOF study only includes predominantly white women so our results are not generalizable to men or other ethnic groups. Third, because the SOF inclusion criteria required the participants to be ambulatory, we could not study the homebound or institutionalized elderly, many of whom might be expected to suffer from hyperkyphosis. Finally, we did not have a measure of back extensor strength that would be a more direct measure of muscle strength than grip strength that could affect posture. Our study also has a number of strengths. It is the first and largest study to incorporate repeated measures of kyphosis over an extended period of 15 years. Second, it had meticulously collected information on vertebral fractures and intervertebral disc morphology. Third, while measurement error in longitudinal assessments of kyphosis is possible, the Cobb angle was ascertained from recumbent films so that postural variability would be less problematic than if the participants had their Cobb angle done in the standing position. Furthermore, we do

not suspect any systematic measurement bias, so that any associated error for any reason would weaken results.

In summary, kyphosis worsens over time in older women and like many geriatric conditions, its progression over 15 years is due to multiple different contributing factors. Besides age, vertebral fractures and low bone density, degenerative disc disease, a family history of hyperkyphosis, concurrent bone density loss, and weight loss are important influences that affect kyphosis progression over time. Of these factors, preventing weight loss, or implicitly, encouraging weight gain (especially lean mass gain), in older persons may prove beneficial. It may be particularly important to prevent muscle mass loss, and randomized controlled trials of exercise interventions targeted at core strengthening are needed. However, although there are non-BMD determinants of hyperkyphosis, the best approach to prevention and treatment may be through slowing bone loss.

Acknowledgments

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Model-Predicted Kyphosis Progression over 15 years
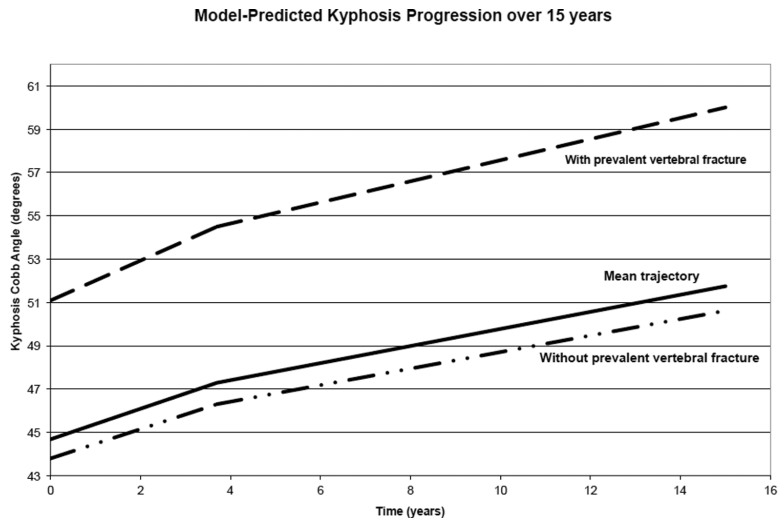
Figure.
Model predicted mean kyphosis progression over 15 years in entire study sample and stratified by prevalent vertebral fracture status

Table 1

Summary of when study measurements completed

Measurements	Baseline (1986–88)	3rd clinic visit (1991)	8th clinic visit (2002-04)
Age	X		
Clinic site	X		
Cobb angle measure of kyphosis	X	X	X
Prevalent vertebral fracture	X		
Disc height	X		
Bone mineral density of the calcaneus	X	X*	
Harpenden stadiometer measure of height	X		
Weight in kilograms	X	X	X
Smoking status (never vs. past/present)	X		
Alcohol use (drinks per week, adjusted for atypical drinking)	X		
Self-reported physical activity (kilojoules/wk/past year)	X		
Gait speed over 6 meters	X	X	X
Grip strength via hand-held isometric dynanometer	X	X	X
Family history of hyperkyphosis	X		
Incident vertebral fracture		X	X

*4th clinic visit, average 3.7 years after baseline film.

Table 2

Baseline Characteristics of the Study Sample and the Overall Parent Cohort

Baseline characteristic	Study Sample (N =980)	Participants not in the analysis (N = 8724)	P-value
Age (yrs)	69.2 ± 3.7	72.0 ± 5.4	<0.0001
Height (cm)	160.4 ± 6.0	159.1 ± 6.0	<0.0001
Weight (kg)	67.5 ± 11.6	67.0 ± 12.6	0.27
Calcaneal BMD (g/cm^2)	0.42 ± 0.08	0.40 ± 0.10	<0.0001
Prevalent vertebral fracture [*]	12.1%	18.9%	<0.0001
Incident vertebral fracture 3.7 years after baseline [†]	3.1% (N = 30)	5.7% (N=359)	0.0006
Incident vertebral fracture between 3.7 & 15 years later [‡]	16.3% (N=145)	15.7% (N=252)	0.70
Family history of hyperkyphosis (% of at least one affected parent)	26.8%	23.4%	0.02
Degenerative disc disease [§]	21.5% (N = 211)	N/A	N/A
Vertebral wedging index	0.89 ± 0.03	N/A	N/A
Disc height ratio	1.13 ± 0.28	N/A	N/A
Grip strength (kg)	23.3 ± 3.9	22.1 ± 4.4	<0.0001
Walking speed (m/s)	1.09 ± 0.19	1.01 ± 0.22	<0.0001
Self-reported health (% of excellent or good)	91.4%	82.2%	<0.0001
Smoking history (% of current)	8.3%	10.2%	0.06
Alcohol use (drinks/wk adj. for atypical drinks)	2.1 ± 4.2	1.9 ± 4.1	0.17

[*] N = 967, N = 8562 because of missing values

[†] N = 976, N = 6262 because of missing values

[‡] N = 892, N = 16088 because of missing values

[§] A disc was defined as having degenerative disc disease (DDD) if the disc wedging ratio was greater than 3 standard deviations below the level-specific sample mean or the disc height compression ratio was less than 0.80, and there was no fracture in the vertebral bodies on either side of the disc.

Table 3

Unadjusted beta coefficients for association between selected characteristics and baseline kyphosis, short-term progression of kyphosis, and long-term progression of kyphosis[*]

Correlate	Contemporaneous Association	Short-term kyphosis progression (average 3.7 yrs)	Long-term kyphosis progression (average 15 yrs)
	β-estimate (95% CI)	β-estimate (95% CI)	β-estimate (95% CI)
Baseline age (yrs)	**0.28 (0.078, 0.48)**	0.01 (−0.06, 0.48)	**0.16 (0.14, 0.31)**
Parental dowager's hump	**3.3 (1.6, 5.0)**	−0.10 (−0.70, 0.58)	0.71 (−0.41, 1.8)
Prevalent vertebral fracture	**8.0 (6.0,10.1)**	**1.1 (0.30, 2.0)**	**3.7 (2.3, 5.0)**
Number of prevalent vertebral fractures	**4.3 (3.2, 5.3)**	0.48 (0.05, 0.92)	**1.8 (1.0, 2.5)**
Vertebral wedging index[†]	**54.8 (51.5, 58.2)**	0.37 (−.23, 1.58)	**4.3 (1.2, 7.5)**
Calcaneal bone mineral density (per 0.1 gm/cm^2)	**−2.4 (−3.3, −1.5)**	0.08 (−0.3, 0.4)	**−1.5 (−2.1, −0.9)**
Degenerative Disc Disease[‡]	**5.1 (3.3, 6.9)**	−0.11 (−0.84,0.6)	**1.4 (0.16, 2.6)**
Disc Height Ratio[†]	**−2.5 (−3.2, −1.8)**	**0.46 (0.19, 0.73)**	0.27 (−0.18, 0.73)
Weight (kg)	0.004 (−0.06, 0.07)	−0.01 (−0.03, 0.02)	−0.04 (−0.08, 0.002)
Maximum grip strength (kg)	−0.19 (−0.39, 0.002)	−.023 (−0.10, 0.06)	−0.04 (−0.17, 0.08)
Walking speed (m/s)	−1.9 (−5.9, 2.1)	1.6 (−0.03, 3.1)	**3.2 (0.60, 5.8)**
Self-reported physical activity (% of excellent or good)	0.0 (−0.0004, 0.0004)	0.0 (−0.0003, 0.0003)	0.0 (−0.004, 0.0001)
Current smoking	**−1.9 (−3.5, −0.37)**	−0.23 (−0.86, 0.40)	−0.65 (−1.7, 0.37)
Alcohol use (drinks/wk adjusted for atypical drinks)	**−1.3 (−1.7, −1.0)**	−0.07 (−0.21, 0.57)	−0.02 (−0.37, 0.07)
Incident vertebral fracture	**4.5 (3.5, 5.5)**	–	–
Increase in calcaneal bone density (per 0.1 gm/cm^2)	**−2.5 (−3.9, −1.0)**	–	–
Increase in weight (kg)	**−0.19 (−0.24, −0.14)**	–	–
Increase in grip strength (kg)	−0.003 (−0.07, 0.07)	–	–

[*] Each row represents a separate model that includes clinic and one predictor. For predictors listed above the grey bar, the contemporaneous association refers to the association with baseline kyphosis. For predictors below the grey bar, the contemporaneous association refers to associations with kyphosis after the change (i.e. incident vertebral fracture, change in bone mineral density, weight, or grip strength). Of note, change in bone mineral density was calculated between baseline and an average of 5.7 years later.

[†] Unit is a difference of per 0.20 in the wedging index or height ratio.

[‡] A disc was defined as having degenerative disc disease (DDD) if the disc wedging ratio was greater than 3 standard deviations below the level-specific sample mean or the disc height compression ratio was less than 0.80, and there was no fracture in the vertebral bodies on either side of the disc.

Table 4

Multivariable-adjusted beta coefficients for association between selected characteristics and baseline kyphosis, short-term kyphosis progression, and long-term kyphosis progression[*]

Correlate	Contemporaneous Association	Short-term kyphosis progression (average 3.7 yrs)	Long-term kyphosis progression (average 15 yrs)
	β-estimate (95% CI)	β-estimate (95% CI)	β-estimate (95% CI)
Baseline age (yrs)	0.17 (−0.03, 0.38)	−0.01 (−0.09, 0.07)	−0.02 (−0.17, 0.13)
Parental dowager's hump	**2.7 (1.03,4.3)**	−0.21 (−0.87, 0.45)	0.24 (−0.81, 1.3)
Prevalent vertebral fracture	**6.0 (3.9, 8.0)**	**0.93 (0.09,1.8)**	**2.4 (1.0,3.8)**
Calcaneal bone density (per 0.1 gm/cm²)	**−2.0 (−3.0 −1.1)**	0.3 (−0.07, 0.70)	**−1.0 (−1.7, −0.4)**
Degenerative Disc Disease [†]	**3.5 (1.7, 5.3)**	0.39 (**−1.1,** 034)	0.16 (−1.1, 1.4)
Weight (kg)	**0.08 (0.01, 0.16)**	**−0.03 (−0.06, 0.001)**	−0.03 (−0.07, 0.02)
Maximum grip strength (kg)	−0.10 (−0.30, 0.11)	−0.005 (−0.09, 0.08)	0.04 (−0.10, 0.18)
Gait speed (m/s)	0.51 (−3.7,4.7)	1.3 (−0.43,3.0)	2.2 (−0.59, 4.9)
Self-reported physical activity (% of excellent or good)	0.0 (−0.0004, 0.0005)	0.0 (−0.0003, 0.0001)	0.0 (−0005, 0.0001)
Current smoking	**−1.9 (−3.5, -0.41)**	−0.05 (−0.67, 0.57)	−0.52 (−1.5, 0.47)
Alcohol use (drinks/wk adjusted for atypical drinks)	0.64 (−1.2, 2.4)	−0.61 (−1.3, 0.12)	0.11 (−1.3, 1.1)
Incident vertebral fracture	**3.8 (2.7, 4.8)**	–	–
Increase in calcaneal bone density (per 0.1 gm/cm²)	**−2.0 (−3.4, −0.49)**		
Increase in weight (kg)	**−0.20 (−0.26, -0.15)**	–	–
Increase in grip strength (kg)	0.0008 (−.08, 0.08)	–	–

[*] All associations are adjusted for other predictors in the table and clinic. For predictors listed above the grey bar, the contemporaneous association refers to the association with baseline kyphosis. For predictors below the grey bar, the contemporaneous association refers to associations with kyphosis after the change (i.e. incident vertebral fracture, change in bone mineral density, weight, or grip strength). Of note, change in bone mineral density was calculated between baseline and an average of 5.7 years later.

[†] A disc was defined as having degenerative disc disease (DDD) if the disc wedging ratio was greater than 3 standard deviations below the level-specific sample mean or the disc height compression ratio was less than 0.80, and there was no fracture in the vertebral bodies on either side of the disc.