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BASIC SCIENCE

Scheuermann Kyphosis in Nonhuman Primates

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Study Design. A cadaveric survey of the thoracic spines of extant species of nonbipedal primates for the presence of Scheuermann kyphosis.

Objective. To determine the presence and prevalence of Scheuermann kyphosis in quadrupedal species of the closest living relatives to humans to demonstrate that bipedalism is not an absolute requirement for the development of Scheuermann kyphosis.

Summary of Background Data. The etiology of Scheuermann kyphosis remains poorly understood. Biomechanical factors associated with upright posture are thought to play a role in the development of the disorder. To date, Scheuermann kyphosis has been described only in humans and extinct species of bipedal hominids.

Methods. Thoracic vertebrae from 92 specimens of *Pan troglodytes* (chimpanzee) and 105 specimens of *Gorilla gorilla* (gorilla) from the Hamann-Todd Osteological Collection at the Cleveland Museum of Natural History were examined for Scheuermann kyphosis on the basis of Sorenson criteria and the presence of anterior vertebral body extensions and for the presence of Schmorl nodes.

Results. Two specimens of *P. troglodytes* (2.2%) were found to have anatomic features consistent with Scheuermann kyphosis including vertebral body wedging greater than 5° at 3 or more adjacent levels and the presence of anterior vertebral body extensions. One of the affected specimens (50%) demonstrated the presence of Schmorl nodes whereas 2 of the unaffected specimens (2.2%) had Schmorl nodes. None of the specimens of *G. gorilla* (0%) were found to have anterior vertebral body extensions characteristic of Scheuermann kyphosis or Schmorl nodes.

Conclusion. Thoracic kyphotic deformity consistent with Scheuermann kyphosis exists in quadrupedal nonhuman primates. Bipedalism is not a strict requirement for the development of Scheuermann kyphosis, and the evolutionary origins of the disease predate the vertebral adaptations of bipedal locomotion.

Key words: Scheuermann, kyphosis, primates, vertebral body extensions, bipedalism. **Spine 2012;37:E1432–E1437**

The etiology of Scheuermann kyphosis remains poorly understood. Previous investigations have pointed toward both genetic and environmental factors contributing to the pathogenesis of the disorder.[1–3] Several studies have shown an increased familial incidence of Scheuermann kyphosis.[1] Recently, a study from the Danish Twin Registry demonstrated a heritability of 74% and concluded that there is a major genetic contribution to the etiology of Scheuermann disease.[3] Mechanical or environmental factors have been proposed on the basis of histopathologic examination of affected vertebral segments and the results of nonoperative treatment modalities.[1] Histopathologic studies of affected individuals have demonstrated disorganized endochondral ossification in the anterior portion of the vertebral endplates similar to that seen in the proximal tibial physis in Blount disease.[2,4,5] The effects of asymmetric mechanical forces on the growth plates of long bones and vertebrae have been suggested to contribute to the development and progression of Blount disease, slipped capital femoral epiphysis, and Scheuermann kyphosis.[6–8] Animal studies have demonstrated that compressive and tensile forces can modulate vertebral body longitudinal growth in agreement with the Hueter-Volkmann law and that asymmetric forces can generate vertebral body wedging.[9,10] Investigations of management of patients with Scheuermann kyphosis with bracing have shown correction of vertebral body wedging, suggesting that correction of mechanical forces can alter the progression of the disorder.[11,12] Although it is possible that the etiology of Scheuermann kyphosis is multifactorial, including both a genetic predisposition and a mechanical contribution, the extent of the influence of these factors is unknown.

To date, Scheuermann kyphosis has not been described in extant species of nonhuman primates. Pathologic features consistent with Scheuermann kyphosis have been described in the fossil remains of the extinct species *Australopithecus afarensis* from specimen AL-288, also known as "Lucy."[13,14] The thoracic vertebrae of AL-288 demonstrate vertebral body wedging as well as anterior vertebral body extensions at multiple levels.[13] The presence of Scheuermann kyphosis in

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A. afarensis, a nonhuman biped, and its absence in other species of nonbipedal primates has been suggested as support for a mechanical origin of the disorder.[2]

Other human spinal disorders with a proposed biomechanical component, such as spondylolysis, idiopathic scoliosis, and vertebral compression fractures, have not been described in wild nonhuman primates.[15] The development of these conditions in humans has been attributed to the structural and mechanical adaptations associated with habitual bipedal locomotion.[16] Spondyloarthropathies, which have a strong genetic component, including skeletal hyperostosis, psoriatic arthritis, and ankylosing spondylitis, have been described in gorilla and other primates.[17–20] Similarly, other spinal disorders associated with genetic factors and not solely explained by the morphologic adaptations of bipedalism may be present yet undiscovered in nonhuman primates.

The aim of our study was to describe the presence of Scheuermann kyphosis in nonbipedal primates to determine whether the mechanical forces associated with bipedalism are a requirement for the development of the disorder. Given evidence that there seems to be a strong genetic component associated with Scheuermann kyphosis, we hypothesized that the disease could be present in nonbipedal primates, chimpanzee and gorilla, closely related to humans.

MATERIALS AND METHODS

The entire collection of nonhuman primates at the Hamann-Todd Osteological Collection at the Cleveland Museum of Natural History was surveyed for specimens of chimpanzee (*Pan troglodytes*) and gorilla (*Gorilla gorilla*) with postcranial elements, which yielded 121 chimpanzee and 185 gorilla specimens. Specimens were excluded from further examination if they were labeled as infant on demographic data, lacked or had incomplete thoracic spinal elements, or were incompletely disarticulated. The remaining specimens, which included 92 chimpanzees and 105 gorillas, underwent further evaluation.

Each thoracic vertebral segment was evaluated qualitatively by 2 observers for the presence of anterior vertebral body extensions, vertebral body wedging, and the presence of Schmorl nodes. Each vertebral segment was then measured quantitatively by a single observer using digital sliding calipers for anterior vertebral body height, posterior vertebral body height, and the maximum anterior to posterior diameter in the midsagittal plane. The degree of vertebral body wedging for each segment was then calculated using the formula determined by Scoles *et al*.[2] Specimens with significant soft-tissue remains that precluded accurate measurement were excluded from quantitative evaluation. Specimens that met Sorenson criteria of 3 or more adjacent vertebral bodies with 5° or more of anterior wedging and the presence of anterior vertebral body extensions were considered to have Scheuermann kyphosis.

Demographic data were collected from the Hamann-Todd Osteological Collection database. The distribution of each individual by species, sex, relative age, and capability to be measured quantitatively can be seen in Table 1. All specimens included in the study were wild caught. Exact specimen ages were unknown, but specimens were categorized as infant, juvenile, or adult. All specimens of chimpanzee were from a single species, *P. troglodytes*, and all gorilla specimens were classified as lowland gorillas. In some cases, the sex of the specimen was not known.

TABLE 1. Demographic and Assessment Data of Study Specimens

	P. troglodytes (Chimpanzee) n (%)	*G. gorilla* (Gorilla) n (%)
Total	92	105
Male	31 (33.7)	69 (65.7)
Female	51 (55.4)	35 (33.3)
Unknown sex	10 (10.9)	1 (1.0)
Adult	62 (67.4)	93 (88.6)
Juvenile	30 (32.6)	12 (11.4)
Quantitative	52 (56.5)	72 (68.6)
Qualitative	40 (43.5)	33 (31.4)
Wild caught	92 (100)	105 (100)

Given that exact specimen ages were not known, and that vertebral body wedging may be secondary to age-related degenerative changes, the entire collection of chimpanzees and gorillas was surveyed for the presence of arthritic changes in the lumbar spine. This survey was performed in conjunction with a concomitant study of primate spinal arthrosis conducted by the authors. Chimpanzee and gorilla specimens with intact lumbar vertebral segments, 63 chimpanzees and 93 gorillas, were examined for the presence of peripheral rim osteophytes in the vertebral endplates at each level, L1–L4, and sacrum. Osteophytic reaction was graded on a scale of 0 to 4 ranging from no arthrosis to complete ankylosis on the basis of the grading scale used previously in human cadaveric studies by Eubanks *et al*.[21]

To assess intraobserver reliability of the measurements taken, repeated measurements of 26 vertebral segments for thoracic specimens and 20 vertebral segments for lumbar specimens were undertaken on 3 separate days with 2-day intervals. Statistical analyses were performed using commercially available software (JMP v5.0; SAS Institute, Inc., Cary, NC) to determine intraobserver reliability using the Pearson product-moment correlation coefficient analysis. The level of significance was set at $P < 0.05$. For thoracic vertebral measurements, the Pearson correlation for intraobserver reliability was 0.99 ($P < 0.001$). The intraobserver reliability for the grade of lumbar osteophytosis was calculated using the multiobserver κ statistic based on intraclass correlations. Analysis of the data demonstrated good intraobserver reliability with κ values from 0.81 to 0.94. Comparisons of the vertebral body wedging angles between affected and unaffected specimens of *P. troglodytes* were made using a Student *t* test.

RESULTS

Two specimens of chimpanzee (*P. troglodytes*) (2.2%) were found to have morphologic features consistent with Scheuermann kyphosis, which included Sorenson's criteria of vertebral body wedging greater than 5° at 3 or more adjacent vertebral body levels and anterior vertebral body extensions. An additional 13 specimens of chimpanzee (14.1%) and 5 specimens of gorilla (4.8%) met Sorenson's criteria but did not exhibit anterior vertebral body extensions. None of the gorilla specimens (0%) met Sorenson's criteria and displayed anterior vertebral body extensions. Table 2 shows the mean calculated degree of vertebral body wedging at each level for gorilla and chimpanzee compared with the affected chimpanzee specimens, HTB 1721 and HTB 1741.

Schmorl nodes were found in 2 (2.2%) of the unaffected chimpanzee specimens and 1 (50%) of the affected chimpanzee specimens. The affected chimp, HTB 1721, demonstrated Schmorl nodes at 5 levels, the superior endplates of T8 and T10–T13 and the inferior endplate of T10. One of the unaffected chimps, HTB 0239, demonstrated Schmorl nodes at 3 levels, superior endplates of T7, T8, and T12, and the other chimp, HTB 1707, had a node at 1 level, superior endplate of T3. There were no Schmorl nodes found in any of the gorilla specimens.

The unaffected chimpanzees that met Sorenson's criteria for vertebral body wedging but did not demonstrate anterior vertebral body extensions had an average of 5.8 levels of significant wedging (wedging >5°), with a range of 3 to 13 levels. Six of the 13 (46.2%) unaffected chimps that met Sorenson's criteria had only 3 levels of significant wedging and involved only levels T11–T13. Five of the unaffected chimps demonstrated significant wedging at 8 or more levels.

Both of the affected chimpanzees were adult females. Both demonstrated 5 adjacent levels of vertebral body wedging greater than 5°. The affected levels were centered at T11 (T9–T13) and T10 (T8–T12) for HTB 1721 and HTB 1741, respectively. For HTB 1721, the level of greatest vertebral body wedging was T12 (11.8°) and for HTB 1741, the level of greatest wedging was T11 (7.4°). Figures 1 through 3 show photographs of vertebral segment T11 from HTB 1721. Figures 4 and 5 show photographs of T10 from HTB 1741. Both specimens demonstrate anterior extension of the vertebral body beyond the rim of the ring apophysis. Figure 2 demonstrates the presence of a Schmorl node in the superior endplate of T11 from HTB 1721.

Both affected chimpanzee specimens, HTB 1721 and HTB 1741, demonstrated no lumbar vertebral osteophytosis (arthrosis grade 0) at all levels, L1–L2, L2–L3, L3–L4, and L4–S1. The overall prevalence of lumbar arthrosis based on the presence of vertebral body osteophytes grade 1 or higher for 1 or more levels was 19.0% for chimpanzees and 31.2% for gorillas.

DISCUSSION

The presence of Scheuermann kyphosis in nonbipedal primates lends support for a genetic origin of the disorder and argues against a purely mechanical etiology. Vertebral bodies of chimpanzee specimens, which meet Sorenson's

TABLE 2. Mean Degree of Vertebral Body Wedging and Standard Deviation of Gorilla and Unaffected Chimpanzee Specimens Compared With Affected Specimens

Thoracic Level	Gorilla Mean Degree (SD)	Pan (Chimp) (Unaffected) Mean Degree (SD)	Pan (Chimp) (HTB 1721) (Affected)	Pan (Chimp) (HTB 1741) (Affected)	*P* Unaffected *vs.* Affected Pan
T1	3.2 (2.7)	5.6* (3.5)	3.0	8.0*	0.506
T2	3.5 (2.0)	5.2* (3.4)	3.5	4.7	0.868
T3	2.3 (2.0)	3.3 (2.0)	1.3	2.9	0.820
T4	2.8 (1.6)	3.3 (2.3)	0.3	4.1	0.659
T5	2.7 (1.7)	3.3 (2.0)	−0.5	4.1	0.680
T6	2.7 (1.8)	3.1 (2.2)	0.2	4.4	0.619
T7	2.9 (1.9)	3.4 (2.6)	0.5	4.8	0.363
T8	2.3 (1.5)	3.0 (1.9)	2.0	5.6*	0.071
T9	4.0 (8.6)	3.6 (2.3)	6.5*	5.1*	0.097
T10	2.1 (1.7)	3.6 (2.4)	8.2*	5.8*	0.047†
T11	3.4 (1.9)	4.6 (2.9)	9.2*	7.4*	0.294
T12	5.7* (6.0)	6.2* (3.4)	11.8*	5.4*	0.381
T13	3.2 (2.1)	5.6* (2.5)	7.4*	4.8	0.506

**Vertebral body wedging greater than 5°. P values represent results of Student t test comparing unaffected chimpanzee specimens with affected chimpanzee specimens.*

†P ≤ 0.05.

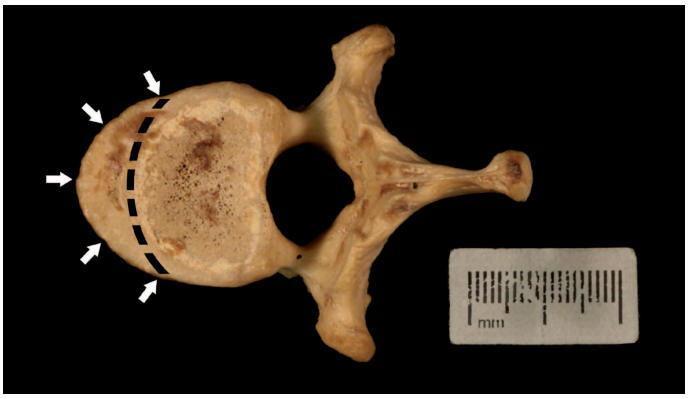

Figure 1. Inferior view of T11 vertebra from affected chimpanzee specimen, HTB 1721, demonstrating anterior vertebral body extension beyond the level of the ring apophysis. Dashed line represents margin of ring apophysis. Arrows highlight anterior vertebral body extension.

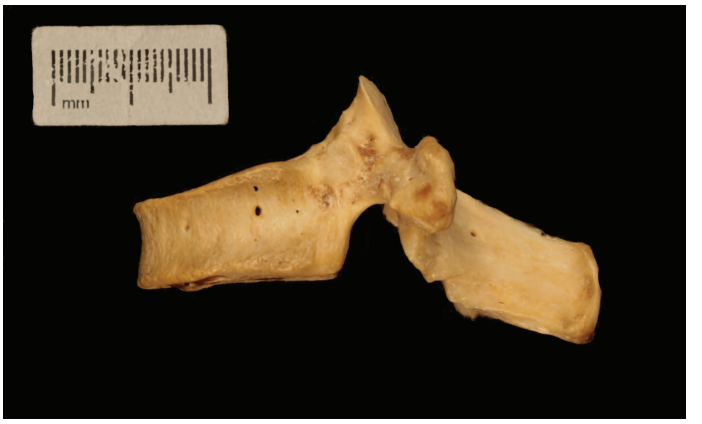

Figure 3. Lateral view of T11 vertebra from affected chimpanzee specimen, HTB 1721, demonstrating vertebral body wedging.

criteria for vertebral body wedging, also demonstrate the characteristic anterior vertebral body extensions seen in Scheuermann kyphosis in humans. The presence of this deformity in the closest living relative of humans, chimpanzees, suggests a contributing genetic factor.[22] Mechanical forces in the thoracic spine of humans and chimpanzees may contribute to the onset and progression of this deformity. However, the forces on the thoracic spine with habitual upright posture and predominately bipedal locomotion are not an absolute requirement for development of Scheuermann kyphosis.

The absence of identification of Scheuermann kyphosis in gorillas has several possible explanations. First, the lack of Scheuermann kyphosis in gorillas in this study does not exclude its presence in this species. It is possible that the prevalence of Scheuermann kyphosis in gorillas may be too low to detect with our sample size. Another possibility is that the differences in posture and locomotion between gorillas and chimps are such that mechanical forces contributing to Scheuermann kyphosis are not present or are present less frequently in gorillas. In general, adult gorillas are considered to exhibit more terrestrial quadrupedalism and less suspensory activities than their more arboreal counterparts, chimpanzees.[23,24] However, when adjusted for body size and ontogeny, because gorillas are larger and mature faster, chimpanzees and gorillas have very similar percentage of time spent in different locomotor activities and sequences of locomotor development.[23] It is possible that the accelerated maturation and transition to predominantly quadrupedal locomotion may account for altered mechanics in the thoracic spine of the developing gorilla; however, this remains unclear.[23] Finally, it is possible that genetic factors influencing the development of Scheuermann kyphosis in humans are present in chimps but absent in gorillas because humans and chimps share a more recent common ancestor.[22] However, although heritability of Scheuermann kyphosis has been demonstrated, specific genetic factors remain unidentified.[3]

There were a significant number of chimpanzee and gorilla specimens, 14.1% and 4.8%, respectively, that

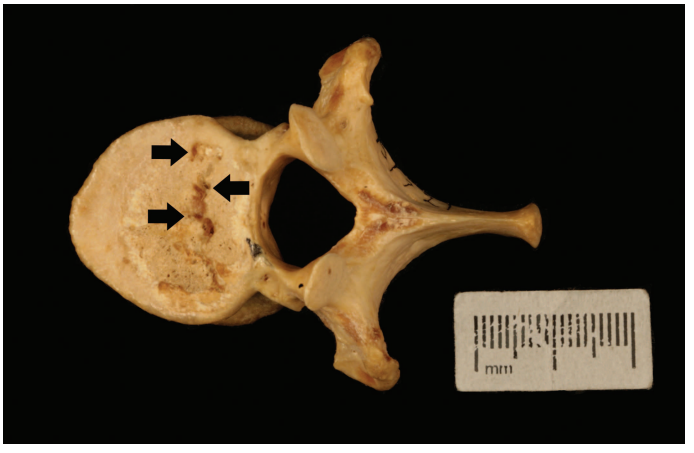

Figure 2. Superior view of T11 vertebra from affected chimpanzee specimen, HTB 1721, demonstrating anterior vertebral body extension and a Schmorl node. Arrows highlight Schmorl node in the superior endplate.

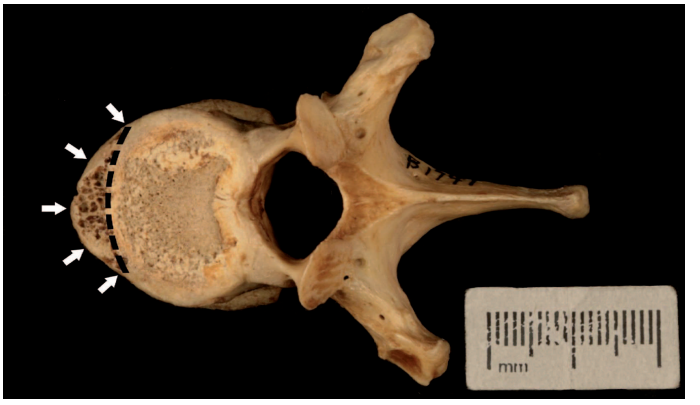

Figure 4. Superior view of T10 vertebra from affected chimpanzee specimen, HTB 1741, demonstrating anterior vertebral body extension. Dashed line represents margin of the ring apophysis. Arrows highlight anterior vertebral body extension.

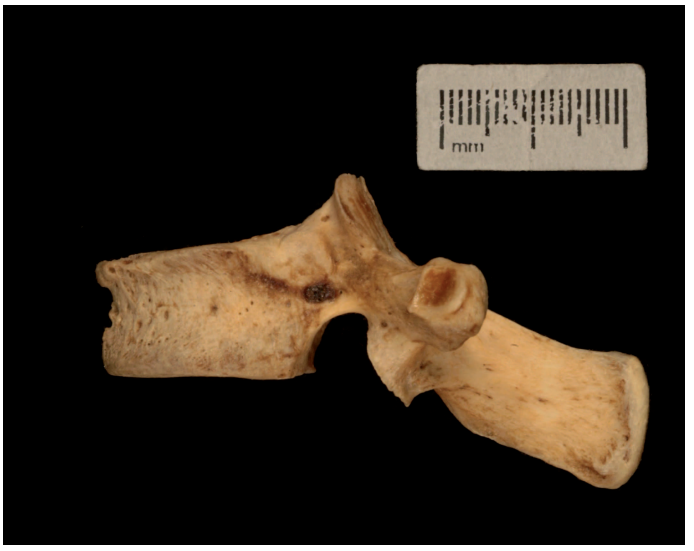

Figure 5. Lateral view of T10 vertebra from affected chimpanzee specimen, HTB 1741, demonstrating vertebral body wedging.

met Sorenson's criteria for vertebral body wedging that were not determined to represent cases of Scheuermann kyphosis. It is possible that these cases represent other causes of kyphosis, such as insufficiency fracture, degenerative disease, or congenital deformity. Low bone mineral density in the osteoporotic range for humans has been reported in chimpanzees, but its occurrence is rare and has not been associated with vertebral compression fractures in wild caught apes.[15,25] Although the exact ages of the chimpanzees and gorillas in our study were not known, they were all wild caught. Wild caught ape populations in general include younger adults than captive ape populations.[26] In addition, we attempted to account for the possibility of age-related degenerative changes contributing to kyphosis by examining our population for the presence of lumbar vertebral body arthrosis. The prevalence of lumbar arthrosis in our population was higher than previously reported in cadaveric surveys of chimpanzees and gorillas; however, previous studies only reported prevalence of arthrosis based on moderate and severe osteophytosis.[26,27] The 2 affected specimens with Scheuermann kyphosis in our study demonstrated no vertebral body osteophytosis at any lumbar level, which lowers the likelihood that the observed pathoanatomical findings in the thoracic vertebrae are due to age-related degenerative changes. It has been noted that in contrast with human and extinct bipedal hominid lumbar vertebrae, which are dorsally wedged, nonbipedal primates have naturally ventrally wedged lumbar vertebrae.[28,29] The observed vertebral wedging that occurred in chimpanzees and gorillas in the absence of Scheuermann kyphosis was largely localized to the lower thoracic levels at the thoracolumbar junction. In addition, the mean vertebral wedging in gorillas and unaffected chimpanzees was greater than 5° at T12 for gorillas and T12 and T13 for unaffected chimps. This may indicate that the observed wedging represents a natural thoracolumbar kyphosis present in some apes.

The prevalence of Scheuermann disease in humans has been reported from 0.4% to 8.3%.[30] Damborg *et al*[3] found the prevalence of Scheuermann disease in humans from the Danish Twin Registry to be 2.8%, which is similar to the prevalence found in chimpanzees (2.2%) in our study. Damborg *et al* also found a higher prevalence in males than in females with a male to female ratio of nearly 2:1.[3] Other studies have shown variable incidences of Scheuermann disease between males and females but the majority suggesting a higher incidence in males.[31] The finding of Scheuermann kyphosis in only female chimpanzees in our study is intriguing, but interpretation is limited due to the presence of only 2 affected specimens. If the prevalence of Scheuermann kyphosis is truly higher in female chimpanzees, it may suggest that differences in developmental or environmental factors between chimps and humans such as mode of locomotion or the carrying of young play a role in the sex difference.

The presence of Scheuermann kyphosis in the extinct bipedal hominid, *A. afarensis*, and the absence of previous description in nonbipedal primates have been used as support for a mechanical etiology of the disorder.[2] Our finding of Scheuermann kyphosis in extant nonbipedal primates, chimpanzees, refutes this contention. However, in their description of the vertebral pathology of specimens of *A. afarensis*, Cook *et al*[13] suggested that other patterns of locomotion and activities, such as climbing, crutch-walking, bridging, and arm-swinging, may induce compressive forces in the thoracic spine contributing to the development of Scheuermann kyphosis. It is possible that similar activities in chimpanzees generate significant asymmetric mechanical stresses in the thoracic spine in the absence of habitual bipedalism.

The etiology of Scheuermann kyphosis is likely multifactorial, with an underlying genetic component predisposing to the influences of mechanical and environmental factors. Mechanical forces acting on the vertebral body can affect the rate of growth in the endplate and may contribute to kyphotic deformity. Our findings refute that upright posture and habitual bipedal locomotion are absolute requirements for the generation of such forces and that the adoption of habitual bipedalism in human ancestors predisposed humans to the development of Scheuermann kyphosis. Whether or not the forces generated by certain behavioral activities in humans and chimpanzees contribute to kyphotic deformity remains unclear. Further investigation into the mechanics of the spine during alternative modes of locomotion in nonbipedal primates may help elucidate the etiology of Scheuermann kyphosis as well as other common spinal disorders.

➢ Key Points

- ❑ The presence of Scheuermann kyphosis in nonbipedal primates argues against a purely mechanical etiology of the disorder.
- ❑ The presence of this deformity in the closest living relative of humans, chimpanzees, suggests a contributing genetic factor.
- ❑ Upright posture and habitual bipedal locomotion are not absolute requirements for the development of Scheuermann kyphosis.
- ❑ The evolutionary origins of Scheuermann kyphosis predate the vertebral adaptations of bipedal locomotion.

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