



The Evolution and Function of Human Lumbar Lordosis Variability

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The Evolution and Function of Human Lumbar Lordosis Variability

A dissertation presented

by

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to

The Department of Human Evolutionary Biology

in partial fulfillment of the requirements

for the degree of

Doctor of Philosophy

in the subject of

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The Evolution and Function of Human Lumbar Lordosis Variability

Abstract

Curvature of the human lower spine, known as lumbar lordosis, was a key adaptation for bipedalism that evolved to balance the upright trunk, but measures of lordosis in modern humans and estimates from the fossil record indicate substantial variability within and among hominin groups. This thesis explores the biomechanical consequences of lordosis variability in modern humans in three studies to understand the evolutionary pressures that have shaped the human lumbar spine.

The first study used magnetic resonance imaging and a postural analysis to test three biomechanical models of lordosis variability. The first model tests the correlation between lordosis and trunk-balancing hip moments; the second tests a beam bending model of lordosis variability; and the third tests an interaction and tradeoff between trunk strength and lumbar flexibility. Results show that hip moments are not associated with lordosis, and the beam model explains 25% of lordosis variation. Lordosis is best explained by the strength-flexibility model, which shows that lumbar flexibility modulates the effects of trunk strength such that stronger backs correlate with greater lordosis and stronger abdominals correlate with reduced lordosis. At the same time, low trunk flexibility is associated with reduced lordosis regardless of trunk strength variations.

The second study examines how lordosis affects the lumbar spine during static axial loading. A weight vest experiment measured changes in lordosis under load. The first hypothesis tests whether lordosis decreases under load to provide stability; the second tests time-dependent variations in lumbar deformations during loading; and the third tests the correlation between lordosis and the spine's resistance to bending. Results show that average lordosis decreases with load, and that straighter spines show less variability in lordosis change during loading. Within a loading trial, straighter spines also tend

to straighten while curved spines tend to become more curved. In addition, lordosis negatively correlates with both bending stiffness and elastic modulus, suggesting that straighter lumbar spines provide greater stability.

The third study used accelerometers mounted on the back to examine how lordosis affects lumbar shock attenuation during barefoot walking and running. The first hypothesis tests the effect of lordosis on shock attenuation; the second tests a viscoelastic model of dynamic lumbar motion; and the third tests how intervertebral disc height affects attenuation. Results show that lordosis has no effect on attenuation during walking but a strong effect during running. In addition, greater attenuation is associated with greater lordosis angular displacements and slower angular displacement velocity. Thicker intervertebral discs are also associated with increased attenuation, but lordosis is a stronger predictor of attenuation when controlling for both discs and lumbar posture. These findings suggest that lordosis increases lumbar shock attenuation during running when dynamic impact forces are highest.

The results of this thesis provide context for interpreting lordosis in fossil hominins and shed light on the etiology of modern lumbar spinal pathology. The first study suggests that trunk geometry passively influences spinal shape. This result may explain why the larger upper bodies of straight-backed Neanderthals, who had deeper rib cages and longer pubic bones, may have experienced higher bending moments acting to reduce lordosis. The second and third studies imply a functional tradeoff underlying evolutionary differences in lumbar curvature. Lordosis is associated with increased lower back mobility and greater lumbar shock attenuation capacity, but curved lumbar spines are also associated with less resistance to bending deformations, less stability, and increased risk of injury due to higher intervertebral shearing forces. This tradeoff has important implications for understanding the selection pressures driving adaptive changes in lumbar lordosis through hominin evolution and perhaps may explain the recent rise in lower back pain prevalence.

Table of Contents

Abstract	iii
Table of Contents	v
List of Figures	vi
List of Tables	viii
Acknowledgements	ix
Chapter 1 – An introduction	1
Chapter 2 – Testing biomechanical models of human lumbar lordosis variability	9
Chapter 3 – Effects of lordosis variability on bending deformations in the human lumbar spine during <i>in vivo</i> axial loading	40
Chapter 4 – Effects of lordosis on shock attenuation in the human lumbar spine during walking and running	76
Chapter 5 – Discussion	109
Appendix 1 – Supplementary Data	134

List of Figures

Chapter 2

Figure 2.1: Sagittal plane schematic of the lumbar spine demonstrating hip and lumbar flexion moments.	12
Figure 2.2: Axial MRI scan at L4-L5 showing outlines of trunk muscles and the lumbar vertebral body.	15
Figure 2.3: Illustration comparing three different lumbar lordosis measurements.	18
Figure 2.4: Associations between lumbar lordosis measurements and the external hip flexor moment arm.	23
Figure 2.5: Associations between lumbar lordosis measurements and the beam bending model prediction.	24
Figure 2.6: A heat map illustration of the strength-flexibility model with and without the interaction effect.	25
Figure 2.7: Partial regression plot showing the effects of abdominal and back muscle CSA on trunk flexibility.	28

Chapter 3

Figure 3.1: Sagittal and transverse plane illustrations of the lumbar spine and variables used in the beam model.	45
Figure 3.2: Individual and average changes in lordosis during loading trials.	58
Figure 3.3: Individual and average changes in lumbar strain during loading trials.	59
Figure 3.4: Time-dependent variations in lordosis within loading trials.	61
Figure 3.5: Three examples of time-dependent changes in lordosis within a loading trial.	62
Figure 3.6: Individual and normalized load-displacement and stress-strain curves.	64
Figure 3.7: Associations between lordosis and both bending stiffness and elastic modulus.	66

Chapter 4

Figure 4.1: Mean static and dynamic lordosis during walking and running.	88
Figure 4.2: Example of dynamic changes in lordosis and filtered acceleration signals during walking and running.	90
Figure 4.3: Power spectra of the resultant low- and mid-back acceleration signals during walking and running.	92
Figure 4.4: Resultant shock attenuation during walking and running compared to static and dynamic lordosis.	93
Figure 4.5: Partial regressions of lordosis angular displacement and displacement velocity vs. shock attenuation.	94
Figure 4.6: Partial regressions between disc height and attenuation during running, controlling for lordosis.	95

Chapter 5

Figure 5.1: Comparison of Cobb and Central Angle lordosis estimates among hominoids.	112
Figure 5.2: Lumbar vertebral body areas for Neanderthals and modern humans in this study.	114
Figure 5.3: Tradeoffs in lordosis variability: lumbar bending compliance vs. shock attenuation during running.	116
Figure 5.4: Lumbar vertebral body wedging angles among hominins and local lordosis changes during loading.	121
Figure 5.5: Association between L1 vertebral body wedging angle and lumbar bending compliance.	123

Appendix 1

Appendix Figure 1.1: Comparison of Standing CA between males and females in this study.	134
Appendix Figure 1.2: Sex differences in Standing CA versus lumbar bending elastic modulus	136
Appendix Figure 1.3: Sex differences in Standing CA versus resultant mean shock attenuation during running.	137

List of Tables

Chapter 2

Table 2.1: Mean and standard deviation of variables.	22
Table 2.2: Correlations between lordosis and beam model variables.	24
Table 2.3: Results of the strength-flexibility model regressions.	27
Table 2.4: Correlations between lordosis and trunk muscle size.	28

Chapter 3

Table 3.1: Glossary of variables and abbreviations from the beam bending model.	44
Table 3.2: Summary of study participant anthropometrics.	51
Table 3.3: Means (s.d.) for applied load masses and lumbar deformation variables across loading trials.	56
Table 3.4: GLMMs testing whether lumbar spines tend to straighten under load.	57
Table 3.5: GLMMs testing the association between mean lordosis and time-dependent changes in lordosis.	60
Table 3.6: GLMMs testing associations between lordosis, lumbar bending stiffness, and elastic modulus.	65

Chapter 4

Table 4.1: Summary of study participant anthropometrics.	88
Table 4.2: Means (s.d.) for the dynamic variables compared between walking and running.	89
Table 4.3: Model testing lordosis angular displacement and displacement velocity versus shock attenuation.	95
Table 4.4: Model testing the effects of maximum disc height and lordosis on running shock attenuation.	96

Appendix 1

Appendix Table 1.1: The main findings from Chapters 2-4 controlling for sex as an additional covariate.	135
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Chapter 1 – An introduction

Why is lordosis so variable?

A defining feature of the human spine is its unique S-shaped profile composed of two distinct curves. While the concave curvature of the upper spine is a characteristic humans share with closely related species such as great apes, the convex forward curvature of the lower spine, known as lumbar lordosis, is uniquely derived in the hominin lineage. Lordosis evolved along with a combination of morphological changes to the hominin axial skeleton including elongation of the lumbar vertebral column, wedging of the vertebral bodies, pelvic tilt, increased spinal mobility, and reduced entrapment of the lower vertebrae between the iliac blades (Shapiro, 1993; Pal and Routal, 1999; Pilbeam, 2004; Lovejoy, 2005; Russo, 2010; Lovejoy and McCollum, 2010; Williams, 2012; Whitcome, 2012; Williams et al., 2016). This suite of adaptations allowed the human lumbar spine to achieve its curved shape to balance the mass of the upright torso stably over two legs.

But many questions remain about the origins of human lumbar lordosis. No one knows precisely when lordosis arose because the complete series of lumbar vertebrae rarely preserve in the fossil record. Hints of lumbar curvature appear in the 4.4-million-year-old fragmentary pelvic remains of *Ardipithecus ramidus*, suggesting that lordosis may have been present in early hominins (Lovejoy et al., 2009). However, direct estimates of lordosis from fossil lumbar vertebrae show that there has been substantial variation in lumbar curvature within populations and among fossil hominins (Been et al., 2012). Lordosis estimates overlapping the modern human range (Cobb angle mean: $51 \pm 11^\circ$ sd; range: $24 - 75^\circ$; 95% CI: $49 - 53^\circ$; $n = 106$) have been reported for fossil specimens attributed to *Australopithecus africanus* ($41 \pm 5^\circ$; 95% CI: $35 - 47^\circ$; $n = 2$), *Homo erectus* (45° ; $n = 1$), and early modern humans ($54 \pm 14^\circ$, 95% CI: $35 - 73^\circ$; $n = 2$). However, Neanderthals ($29 \pm 4^\circ$, 95% CI: $24 - 34^\circ$; $n = 3$) and

possibly *Homo heidelbergensis* (Bonmati et al., 2010) appear to have had straighter lumbar spines at the extreme low range of modern variation (Been et al., 2012).

High variability in lumbar lordosis presents the following conundrum: if sufficient lordosis is necessary for maintaining postural balance, then why is it so variable? What drove variations in lordosis through hominin evolution, and how do these variations affect spinal function? Do lordosis variations serve an adaptive purpose? Answering these questions requires an understanding of the potential tradeoffs involved. On the one hand, lordosis not only helps maintain postural balance—especially during pregnancy (Whitcome et al., 2007)—but also may increase lower back mobility allowing the lumbar spine to serve as “shock absorber” to cope with impact-related accelerations generated by the foot’s collision with the ground during running (Rak, 1993; Adams et al., 2006). However, few studies have measured the influence of lumbar lordosis on shock accelerations propagated through the spine during locomotion. On the other hand, the benefits of a curved lumbar posture may come at the costs of reduced mechanical stability of the spine as a support structure while also increasing the risk of injury due to higher levels of intervertebral shearing forces (Scholten et al., 1988; Aspden, 1989; Shiraz-Adl et al., 2002; Arjmand and Shirazi-Adl, 2005; Ward and Latimer, 2005; Adams et al., 2006; Meakin et al., 2008; Castillo and Lieberman, 2015). Intervertebral shearing arises due to an increase in non-normal forces acting between adjacent vertebrae as a function of the angle of inclination of vertebrae relative to the horizontal. Although straighter lumbar spines are assumed to provide greater resistance to loading and lower intervertebral shearing, little is known about how natural variations in lordosis influence the lumbar spine’s bending stiffness during loading. This thesis explores the tradeoffs underlying differences in lumbar curvature in order to understand the evolutionary origins and adaptive significance of lordosis variability.

Thesis summary

Two main questions are investigated in this thesis: what drives variations in lordosis, and what are the functional consequences of these variations? These two questions were tested on the same sample of study participants across three different experiments (Chapters 2-4). In summary, Chapter 2 addresses the first question of what drives lordosis variability by testing biomechanical factors that have been hypothesized to explain differences in natural standing lordosis. The second question of the functional consequences of lordosis variability is addressed in Chapters 3 and 4, which examine how variations in lordosis affect the behavior of the lumbar spine during static and dynamic loading. Specifically, Chapter 3 tests a curved beam model of the lumbar spine to illuminate how differences in spinal curvature affect patterns of bending deformations *in vivo* during static axial loading. Chapter 4 investigates how impact shocks are transmitted through the lumbar spine during walking and running in order to test the relationship between variations in lordosis and lumbar shock attenuation.

Several factors are known to correlate with lordosis including age, sex, height, and body mass; postural parameters like pelvic tilt and thoracic kyphosis; and environmental effects like occupational loading and physical fitness (Been and Kalichman, 2014). However, these factors tend to covary and offer little explanation for the underlying causes of lordosis variability. For these reasons, Chapter 2 tests three biomechanical models that have been previously proposed to explain lumbar curvature. Using magnetic resonance imaging (MRI) and 3-D postural analyses of participants standing at rest, the first model tests the prediction that lordosis evolved to minimize external hip moments required to balance and stabilize the trunk. The second model tests the prediction that the lumbar spine behaves like a loaded beam, which deforms in a predictable way based on the beam's cross-sectional area, length, and external bending moments. The third hypothesis tests an interaction between trunk muscle strength and sagittal lumbar range of motion, predicting that relatively stronger abdominal muscles and reduced

lumbar flexibility associate with reduced lordosis. Results show that external hip moments are not associated with lordosis, but the beam model explains 25% of the variation in standing lordosis with individual variables of lumbar spine length, lumbar vertebral body cross-sectional area, and bending moments independently associated with reduced lordotic curvature. The strength-flexibility interaction model explains 65% of the variation in standing lordosis, showing that relative trunk strength has a strong influence on lumbar lordosis variability among individuals with greater lumbar flexibility, but among individuals with immobile lumbar spines, lordosis tends to be low regardless of trunk strength variations. Together these findings imply that some variations in lordosis are due to upper body geometry and mass, but soft tissue properties affecting trunk muscle strength and passive sagittal lumbar compliance are also important predictors of lordosis.

Chapter 3 investigates the functional consequences of variations in lordosis during static axial spinal loading. Previous modeling studies and *ex vivo* lab experiments on isolated cadaveric spines have shown that variations in lumbar posture affect patterns of deformation under axially applied loads (Adams and Hutton, 1985; Scholten et al., 1988; Aspden, 1989; Patwardhan et al., 1999; Shirazi-Adl and Parnianpour, 1999; Shirazi-Adl et al., 2002). However, few studies have tested how the lumbar spine deforms *in vivo* (e.g. El-rich et al., 2004; Meakin et al., 2008; Rodriguez-Soto et al., 2013), thus limiting our ability to estimate the lumbar spine's overall resistance to bending forces. A combination of beam modeling predictions, MRI imaging, and weight vest loading experiments are used to study lumbar bending stiffness *in vivo*. Three main hypotheses are tested. The first hypothesis is that average lordosis decreases during axial loading to provide greater stability. The second hypothesis is that lumbar posture affects time-dependent patterns of bending deformation during loading such that lordosis correlates with variability in lumbar posture during loading. The third hypothesis is that variations in unloaded lordosis predict lumbar bending stiffness and elastic modulus such that naturally straighter spines show greater resistance to deformation. Results show that the average lumbar spine tends to straighten

during loading, and straighter spines are more stable under load, showing less time-dependent variability of lordosis change during a loading trial. In addition, relative changes in lordosis follow a pattern in which straighter lumbar spines tend to straighten and more curved spines tend to become more curved after two-minutes of loading. Furthermore, bending stiffness and elastic modulus of the lumbar spine are inversely associated with unloaded lordosis, which explains 52% and 64% of the variation in lumbar spinal bending stiffness and elastic modulus, respectively. These results collectively suggest that naturally straighter lumbar spines are more stable and have greater resistance to bending deformations in response to external carried loads.

Chapter 4 explores how variations in lordosis affect dynamic shock accelerations in the lumbar spine during walking and running. Collisions between the foot and ground during locomotion generate impact-related shock waves that propagate up through the body to the head. Without mechanisms for attenuation, these high levels of acceleration can potentially cause vestibulo-ocular disturbance and even injury (Pozzo et al. 1990, 1991; Whittle 1999; Davis et al., 2016). Mechanisms for shock attenuation in the lower limb—including both active attenuation due to limb kinematics and passive attenuation due to substrate stiffness and footwear—have been well studied. However, little is known about how shock accelerations propagate through the spine. Using two tri-axial accelerometers mounted on the skin overlying the thoracolumbar and lumbosacral joints during walking and running, Chapter 4 tests three hypotheses about the relationship between lordosis and lumbar shock attenuation. The first hypothesis is that more curved spines show increased spinal shock attenuation while straighter spines are less capable of attenuating impact shocks. The second hypothesis is that the dynamic motion of the lumbar spine reflects viscoelastic properties to attenuate shocks. A viscoelastic beam model is used to predict that higher levels of shock attenuation during running are explained by the overall amplitude of lordosis change and the rate of lordosis change. The third hypothesis tests how the correlated effects of intervertebral disc height (measured from sagittal MRI) and lordosis affect lumbar shock attenuation

when running. Results reveal that variations in both static and dynamic lordosis have no effect on lumbar shock attenuation during walking but have a strong effect during running explaining 30% of the variation in shock attenuation. Furthermore, dynamic changes in lordosis during running support the predicted pattern of viscoelastic behavior such that greater shock attenuation is observed among lumbar spines that have greater amounts of lordosis angular displacement and slower rates of angular displacement. The viscoelastic model explains 41% of the variation in shock attenuation. In addition, results support the hypothesis that thicker intervertebral discs are associated with increased shock attenuation, but when controlling for covariation between disc height and lumbar spinal shape, standing lordosis is a stronger predictor of shock attenuation than discs. Collectively, these findings suggest that individuals with high degrees of lumbar lordosis have greater capacity for shock attenuation in order to reduce dynamic impacts during running but not walking.

The last chapter summarizes the major findings from these three studies and discusses their implications within the broader context of human evolution. Findings are synthesized to advance a new perspective for interpreting variations in lordosis among fossil hominins and to hypothesize about their implications for lumbar spinal pathology today. The costs and benefits of variations in lumbar lordosis are weighed, and fossil data on hominin lumbar vertebral morphology are combined with experimental results to hypothesize about the functional tradeoffs driving variations in the evolution of lumbar spinal posture, particularly in straight-backed Neanderthals. Finally, the implications of these results for modern health are discussed, including how recent changes in human behavior such as reduced physical activity may affect global variations in, causes of, and potential treatments for lumbar pathology.

References

- Adams MA, Hutton WC. 1985. The effect of posture on the lumbar spine. *J Bone Joint Surg* 67(4): 625-9.
- Adams MA, Bogduk N, Burton AK, Dolan P. 2006. The biomechanics of back pain. Edinburgh: Churchill Livingstone.
- Arjmand N, Shirazi-Adl A. 2005. Biomechanics of changes in lumbar posture in static lifting. *Spine* 30(23): 2637-48.
- Aspden RM. 1989. The spine as an arch: a new mathematical model. *Spine* 14(3): 266-74.
- Been E, Gómez-Olivencia A, Kramer PA. 2012. Lumbar lordosis of extinct hominins. *Am J Phys Anthropol* 147: 64–77.
- Been E, Kalichman L. 2014. Lumbar lordosis. *Spine J* 14: 87–97.
- Bonmatí A, Gómez-Olivencia A, Arsuaga JL, Carretero JM, Gracia A, Martínez I, Lorenzo C, de Castro JM, Carbonell E. 2010. Middle Pleistocene lower back and pelvis from an aged human individual from the Sima de los Huesos site, Spain. *Proc Natl Acad Sci USA* 107(43): 18386-91.
- Castillo ER, Lieberman DE. 2015. Lower back pain. *Evol Med Public Health* 1: 2-3.
- Davis IS, Bowser BJ, Mullineaux DR. 2016. Greater vertical impact loading in female runners with medically diagnosed injuries: a prospective investigation. *Br J Sports Med* 50: 887-92.
- El-Rich M, Shirazi-Adl A, Arjmand N. 2004. Muscle activity, internal loads, and stability of the human spine in standing postures: combined model and in vivo studies. *Spine* 29(23): 2633-42.
- Lovejoy CO. 2005. The natural history of human gait and posture: Part 1. Spine and pelvis. *Gait Posture* 21(1): 95-112.
- Lovejoy CO, McCollum MA. 2010. Spinopelvic pathways to bipedality: why no hominids ever relied on a bent-hip-bent-knee gait. *Philos Trans R Soc Lond B Biol Sci* 365: 3289–99.
- Lovejoy CO, Suwa G, Simpson SW, Matternes JH, White TD. 2009. The Great Divides: *Ardipithecus ramidus* reveals the postcrania of our last common ancestors with african apes. *Science* 326: 73-106.
- Meakin JR, Smith FW, Gilbert FJ, Aspden RM. 2008. The effect of axial load on the sagittal plane curvature of the upright human spine in vivo. *J Biomech* 41: 2850–4.
- Pal GP, Routal RV. 1999. Mechanism of change in the orientation of the articular process of the zygapophyseal joint at the thoracolumbar junction. *J Anat* 195(02): 199-209.
- Patwardhan AG, Havey RM, Meade KP, Lee B, Dunlap B. 1999. A follower load increases the load-carrying capacity of the lumbar spine in compression. *Spine* 24(10): 1003-9.
- Pilbeam D. 2004. The anthropoid postcranial axial skeleton: Comments on development, variation, and evolution. *J Exp Zool B Mol Dev Evol* 302B(3): 241-67.
- Pozzo T, Berthoz A, Lefort L, Vitte E. 1991. Head stabilization during various locomotor tasks in humans. II. Patients with bilateral peripheral vestibular deficits. *Exp Brain Res* 85(1) :208–17.
- Pozzo T, Berthoz A, Lefort L. 1990. Head stabilization during various locomotor tasks in humans. I. Normal subjects. *Exp Brain Res* 82(1): 97–106.

- Rak Y. 1993. Morphological variation in *Homo neanderthalensis* and *Homo sapiens* in the Levant: a biographic model. In: KimbelKimbel WH, Martin LB, editors. *Species, species concepts, and primate evolution*. New York: Plenum Press. p 523–36.
- Rodriguez-Soto AE, Jaworski R, Jensen A, Niederberger B, Hargens AR, Frank LR, Kelly KR, Ward SR. 2013. Effect of load carriage on lumbar spine kinematics. *Spine* 38(13): E783-91.
- Russo GA. 2010. Prezygapophyseal articular facet shape in the catarrhine thoracolumbar vertebral column. *Am J Phys Anthropol* 142(4): 600-12.
- Scholten PJ, Veldhuizen AG, Grootenboer HJ. 1988. Stability of the human spine: a biomechanical study. *Clin Biomech* 3(1): 27-33.
- Shapiro LJ. 1993. Functional morphology of the vertebral column in primates. In: Gebo DL, editor. *Postcranial adaptation in nonhuman primates*. DeKalb (IL): Northern Illinois University Press. p 121-49.
- Shirazi-Adl A, Parnianpour M. 1999. Effect of changes in lordosis on mechanics of the lumbar spine-lumbar curvature in lifting. *J Spinal Disord* 12(5): 436-47.
- Shirazi-Adl A, Sadouk S, Parnianpour M, Pop D, El-Rich M. 2002. Muscle force evaluation and the role of posture in human lumbar spine under compression. *Eur Spine J* 11(6): 519-26.
- Ward CV, Latimer B. 2005. Human evolution and the development of spondylolysis. *Spine* 30(16): 1808-14.
- Whitcome KK. 2012. Functional implications of variation in lumbar vertebral count among hominins. *J Hum Evol* 62: 486–97.
- Whittle MW. 1999. Generation and attenuation of transient impulsive forces beneath the foot: a review. *Gait Posture* 10(3): 264-75.
- Williams SA, Middleton ER, Villamil CI, Shattuck MR. 2016. Vertebral numbers and human evolution. *Am J Phys Anthropol* 159(S61): S19-S36.
- Williams SA. 2012. Placement of the diaphragmatic vertebra in catarrhines: implications for the evolution of dorsostability in hominoids and bipedalism in hominins. *Am J Phys Anthropol* 148(1): 111-22.

Chapter 2 – Testing biomechanical models of human lumbar lordosis variability

Introduction

The human lumbar spine exhibits several adaptations to cope with the biomechanical demands of bipedal locomotion and orthograde posture. Compared to closely related living species, the human lumbar spine is relatively longer and contains more vertebral elements with a modal pattern of five functional lumbar vertebrae compared to three or four in most great apes (Pilbeam, 2004; Williams et al., 2016). Elongation of the human lumbar spine provides more trunk mobility aided in part by having a greater number of vertebrae with sagittally oriented zygapophyses (rather than coronally oriented zygapophyses in the thoracic spine) and reduced “entrapment” of the caudal-most lumbar vertebrae between the iliac blades, a feature present in great apes (Shapiro, 1993; Pal and Routal, 1999; Lovejoy, 2005; Lovejoy and McCollum, 2010; Russo, 2010; Williams, 2012; Whitcome, 2012). Increased mobility of the human lower back also decouples motions of the thorax and pelvis and permits independent counter-rotation of the upper and lower body during locomotion (Bramble and Lieberman, 2004; Thompson et al., 2015). Additionally, the wedge-shaped profile of lumbar vertebral bodies and intervertebral discs, along with increased lumbar length and flexibility, allow the human lumbar spine to assume a convex ventral curvature known as lordosis.

Although lumbar lordosis (LL) is an adaptation for upright posture, it has both costs and benefits. Lordosis allows hominins to maintain a stable, upright orientation of the trunk at the expense of biomechanical vulnerabilities that may increase our susceptibility to lower back pain and injury (Ward and Latimer, 2005; Whitcome et al., 2007; Castillo and Lieberman, 2015). However, LL varies substantially in living populations and among fossil hominins. Within a modern population, LL measured from standing radiographs can range between 14° and 69° (Vialle et al., 2005). From the fossil record, skeletal estimates (mean $\pm$ s.d.) of lumbar curvature in *Australopithecus africanus* ($41 \pm 4^\circ$; $n = 2$), *Homo*

erectus (45° , $n = 1$), and early modern humans ($54 \pm 14^\circ$, $n = 2$) all overlap modern human LL variation ($51 \pm 11^\circ$, $n = 106$; Been et al., 2012). Yet Neanderthals ($29 \pm 4^\circ$, $n = 3$) and possibly *Homo heidelbergensis* (Bonmati et al., 2010) may have had straighter lumbar spines at the lower range of modern variation (Been et al., 2012). Lumbar lordosis is also clinically relevant because curvature variations affect sagittal postural balance and function and may be linked to pathologies such as spondylolisthesis, osteoarthritis, and disc degeneration (Berlemann et al., 1999; Umehara et al., 2000; Kumar et al., 2001; Chen et al., 2009). Given its evolutionary and biomechanical importance, why is LL so variable?

Understanding the structural and functional bases for lordosis variation is a challenge because the alignment of the spine and balance of the upper body are influenced by several inter-related factors. For instance, LL is mechanically linked to other spinal postural parameters such as thoracic kyphosis, pelvic orientation, and sacral incidence (Vialle et al., 2005; Bailey et al., 2016). Several factors have also been shown to be associated with LL including individual factors such as age, sex, height, and body mass; genetic factors influencing heritability; and environmental factors such as occupational loading, athletic training, and physical fitness (for review, see Been and Kalichman, 2014). However, variables commonly associated with LL, such as physical fitness and muscle strength, are often vaguely defined with little or no mechanistic explanation for associations. Furthermore, many factors known to affect LL tend to covary, such as sex and body size, complicating efforts to test causal relationships.

To better explain inter-individual variations in LL, this study tests three biomechanical hypotheses—none of them mutually exclusive—that have been previously proposed. The first hypothesis (**H1**) almost always cited in the evolutionary literature is that LL is an adaptation for bipedalism that minimizes hip muscle moments required to balance and stabilize the trunk by repositioning the center of mass of the upper body (i.e. head, arms, and trunk; COM_{HAT}) over the lower

limb, thus reducing the mechanical and metabolic costs associated with upright posture (Steindler, 1997; Abitbol, 1988; Lovejoy, 2005; Whitcome et al., 2007; Saha et al., 2007, 2008; Lovejoy and McCollum, 2010). If lordosis is an adaptation for minimizing external hip flexor moments (M_{hip}), then **H1** predicts that LL is negatively correlated with the M_{hip} moment arm, measured as the antero-posterior (A-P) position of COM_{HAT} relative to the hip's center of rotation (Figure 2.1).

The second hypothesis (**H2**) is that the mass of the upper body passively loads the lumbar spine leading to lordotic deformations as predicted by a loaded column or beam. To describe the overall shape of the human thoracolumbar spine, Euler buckling models are frequently used to predict the relationship between spinal curves and loads acting on the vertebral column (Whitcome, 2012; Meakin et al., 1996, 2008). However, experimental studies in other vertebrates often rely on multi-jointed beam bending models to predict spinal deformations in response to loading (Gal, 1993; Long et al., 1997; Etnier, 2001). To test the beam model in humans, the position of COM_{HAT} , which is located anterior to the lumbar spine in standing posture, is assumed to cause bending moments to flex the lumbar spine anteriorly (White and Panjabi, 1978). The second hypothesis therefore predicts that variables associated with the beam model (e.g., beam length, vertebral body cross-sectional area, and bending moments) negatively correlate with LL (Figure 2.1).

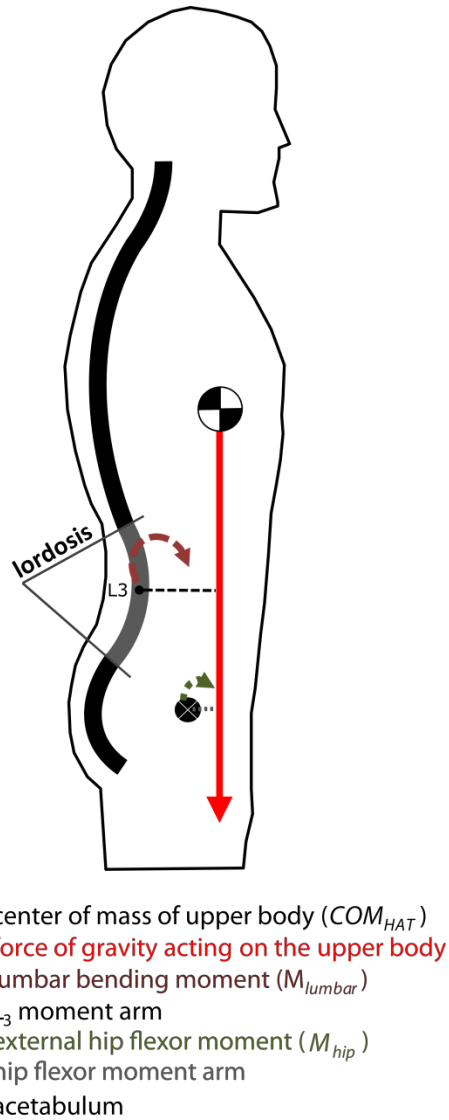


Figure 2.1: Sagittal plane schematic demonstrating the biomechanical variables tested by **H1** and **H2**.

The final hypothesis (**H3**) is that LL variations are driven by an interaction between relative trunk strength (RTS) and sagittal lumbar range of motion (ROM). The hypaxial (abdominal) and epaxial (back) muscles actively support and stabilize the trunk (Wagner et al., 2012), but the relative contributions of these antagonistic muscle groups play a crucial role to affect variations in lordotic curvature. Previous work has shown that individuals with relatively stronger abdominal muscles tend to have reduced LL and less anterior pelvic tilt, while individuals with relatively weaker abdominals have higher LL and increased

anterior pelvic tilt (Walker et al., 1987). Furthermore, the ratio of trunk flexor versus extensor torque negatively correlates with LL (Kim et al., 2005). However, little is known about how spinal mobility interacts with muscle strength to affect LL. Stronger muscles tend to increase joint stiffness, while increased ROM is often associated with greater compliance of ligamentous tissues and passive elastic components of musculotendinous structures. But whether these factors interact and potentially trade off to influence spinal curvature has not been tested. When accounting for the individual and interacting effects of both hypaxial and epaxial muscle size in a multiple regression model against ROM, lumbar ROM is predicted to vary inversely with epaxial muscle size, while increased epaxial size is predicted to increase joint stiffness. Thus, **H3** is that LL variation is explained by an interaction and tradeoff between RTS and ROM.

Materials and Methods

Participants

To minimize potential age-related changes in spinal alignment (Schwab et al., 2006), a cross-sectional sample of young adults (aged 18-35 years) was recruited. Volunteers from the Boston area completed a health questionnaire and were excluded if they had a history of chronic back pain, sciatica, scoliosis, major illness, or injury in the past three months. Written consent was given prior to participation in the study, which was approved by the Committee on the Use of Human Subjects (Harvard University). Protocols were conducted at the same time in the afternoon (between 3-6 PM) to minimize the effects of circadian patterns of the viscoelastic creep in spinal tissues (Strickland and Shearin, 1972; Whitehouse et al., 1974; Wainwright, 1982; Lampl, 1992; Botsford et al., 1994; Voss and Bailey, 1997; Tillman and Clayton, 2001). Thirty participants (15 males, 15 females) completed the study. Mean age was 22.8 ± 4.2 years. The study sample included a diversity of heights (range: 1.54 - 1.88 m;

mean: 1.72 ± 0.09 m) and body masses (range: 45.6 - 89.5 kg; mean: 65.6 ± 11.4 kg). Average body mass index was 22.0 ± 2.5 kg/m² (Table 2.1).

Imaging

Participants were scanned using magnetic resonance imaging (MRI) at the Center for Brain Science Neuroimaging Facility (Harvard University) with a Siemens TIM Trio (3-T) scanner using a standard spine-array and large-flex coils. Participants were scanned in the clinical standard of a neutral, supine position (legs extended, arms resting at their sides). After a midsagittal, single-slice “localizer” scan (TR = 8.6 ms, TE = 4 ms; 7 mm thickness, 1.7 mm/pixel), an axial sequence of 50 transverse slices was acquired from approximately T10 through S2 using the Fast Low Angle Shot (FLASH) imaging protocol (TR = 7.4 ms, TE = 4 ms; 5 mm thickness, 1.44 mm/pixel) during which participants were asked to hold their breath for approximately 30 seconds to reduce diaphragm-related image distortions.

Scans were analyzed using ImageJ (National Institutes of Health). The midsagittal localizer scan was used to measure LL (see below). Axial scans were used to measure cross-sectional areas (CSA) by outlining the borders of the lumbar vertebral bodies and the major anterior and posterior intrinsic trunk muscles that support and stabilize the lumbar spine in the sagittal plane: erector spinae (ES), multifidus (MF), quadratus lumborum (QL), and rectus abdominus (RA) (Figure 2.2). Muscle CSA was measured at the L4-L5 intervertebral level. Cross-sectional areas were quantified as the mean area of three tracings. Intra-observer error was found to be low at 3.6%, measured as the mean coefficient of variation (CV) for the area tracings. Since axial scans are usually non-orthogonal to the trunk muscle lines of action, muscle CSA was corrected for muscle fiber orientation following McGill et al. (1993) by computing the dot product between the scan plane’s perpendicular axis and the unit vector cosine of muscle fiber orientation obtained from the literature (Macintosh and Bogduk, 1986, 1991; Dumas et al., 1991).

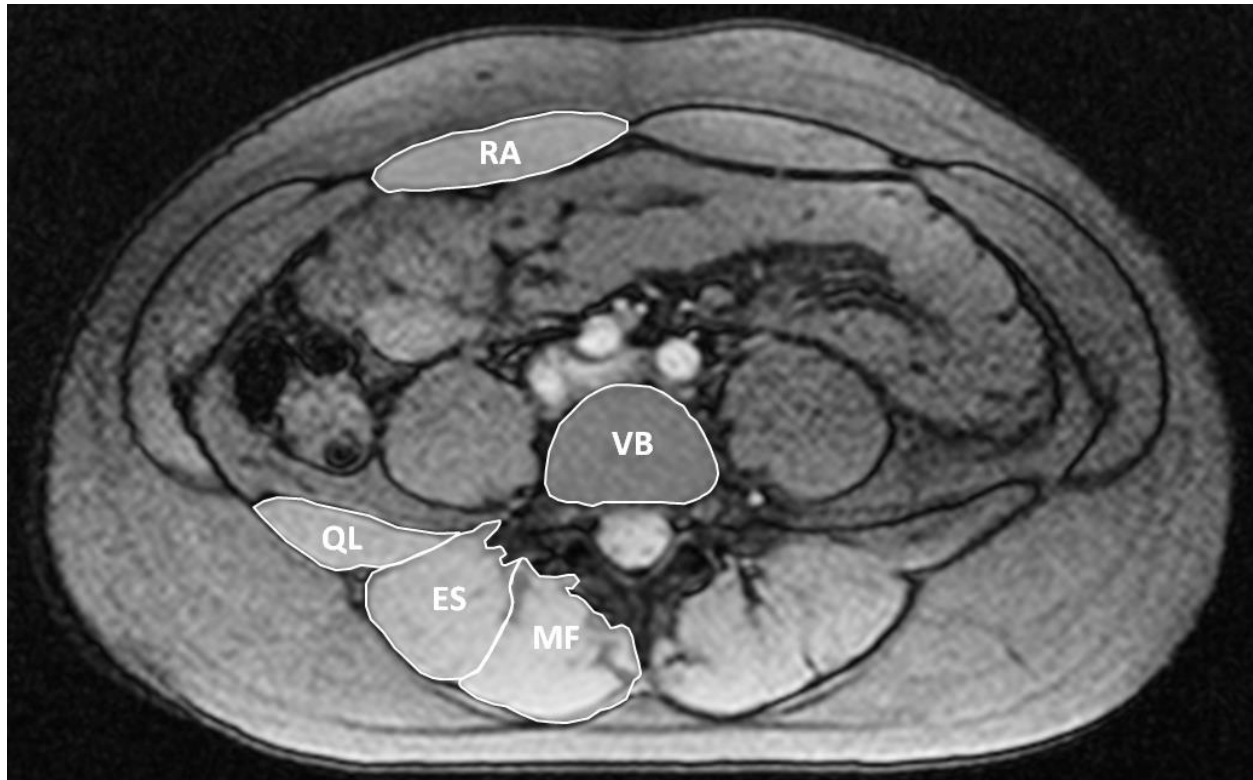


Figure 2.2: Axial MRI scan at the L4-L5 vertebral level showing outlines the rectus abdominus (RA), quadratus lumborum (QL), erector spinae (ES), and multifidus (MF) used to measure cross-sectional areas (CSA). CSA of the L5 vertebral body (VB) is also represented. Both left and right sides were included in the analysis, but muscle outlines are shown on only one side for comparison.

Posture analysis

Within 45 days of the MRI, a posture analysis was conducted. Pairs of retroreflective markers were placed bilaterally on the greater trochanter, anterior and posterior superior iliac spines, iliac crest, acromion process, medial and lateral humeral epicondyles, ulnar and radial styloid processes, and mandibular process. Markers were also placed on the sternal notch and the spinous processes of C7 and T12 through S1. Participants were instructed to stand using a comfortable posture for 2 minutes. Three-dimensional kinematics were collected at 200 Hz using an 8-camera infrared motion capture system (Oqus 1 Series, Gothenburg, Sweden) and Qualysis tracking software (v.2.10).

The position of COM_{HAT} was estimated using Visual3D (C-Motion, v.5). Body segment masses and COM positions were calculated from regression equations following Dempster (1955), and segment inertial properties were computed following Hanavan (1964). These widely used and well-validated methods have been shown to estimate body segment properties reliably in a stationary, standing posture. Though several methods exist for estimating body segment properties, differences between methods are most pronounced for dynamic studies (Rao et al., 2006). Static estimates of body segment COM locations show only small differences between models. For individuals with normal body mass indices, the position of the upper body's COM relative to the hip calculated using the Dempster method differs from the De Leva (1996) method by only 3%, and De Leva estimates of A-P upper body inertial properties correlate strongly with the Hanavan model ($r = 0.90$; Damavandi, 2008).

Measuring lumbar range of motion

Lumbar ROM in the sagittal plane was measured immediately following the posture analysis. Participants were asked to maximally flex the trunk from a standing position with arms outstretched as if bending to touch their toes. They were then asked to stand with hands on the sides of their heads, arching the back in maximum spinal extension (hyperlordosis). Spinal curvature was measured at maximum flexion and extension. Because spinal markers were sometimes obscured from camera view, ROM was measured using an iGaging digital protractor following a technique similar to the surface pantograph method, which correlates with radiographic measurements of LL ($r = 0.80$; Willner, 1981). Range of motion was calculated as the angle difference between maximum flexion and extension (Table 2.1). The average of three measurements was used, and intra-observer error showed low CVs of 2.7%.

Measuring lumbar lordosis

Because there are many methods for quantifying LL (see Vrtovec et al., 2009), this study compared three different LL measurements (Figure 2.3). First, Cobb's Lordotic Angle (Cobb LA) was measured from the midsagittal MRI scans as the intersection between lines drawn along the cranial endplates of L1 and S1. Although often considered the "gold standard" for measuring LL, Cobb LA can fail to capture lordosis variability because different spinal curves can share similar orientations between the L1 and S1 endplates (Been and Kalichman, 2012). Therefore, a Central Angle (CA) method was also used to measure LL, calculated as the best-fit least-squares circle that passes through the 7 points representing T12 through S1. The CA method measures curvature as angle θ such that:

Equation 1
$$\theta = \frac{L}{r}$$

where L is the arc length, and r is the circle's radius. The CA method offers several advantages over Cobb LA because it captures more LL variation and quantifies a size-corrected measure of curvature. Additionally, the CA method can be integrated with beam models that relate curvature to angular deformations (see below). Thus, Supine CA was calculated from the MRI by fitting a circle to the posterior-central borders of the T12-S1 vertebral bodies. Standing CA was found by computing the 2-min average best-fit circle through the T12-S1 spinous process markers from the posture analysis.

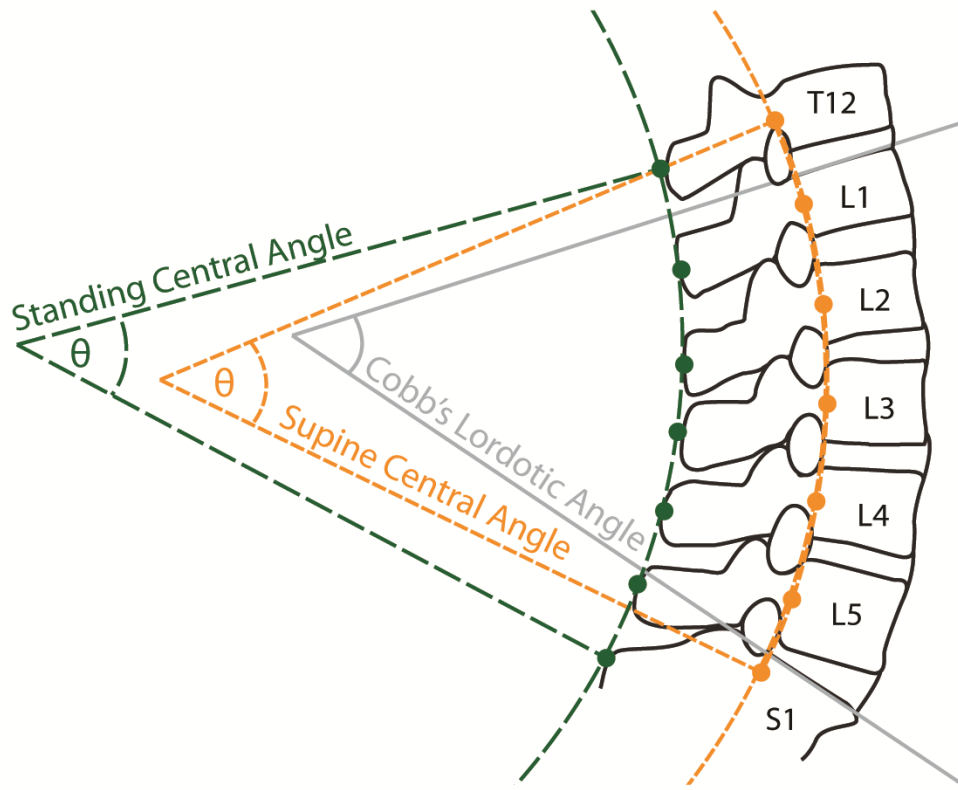


Figure 2.3: Illustration of lumbar lordosis measurements compared in this study: Cobb's Lordotic Angle (Cobb LA), Supine Central Angle (Supine CA), and Standing Central Angle (Standing CA).

Hypothesis testing

To test **H1**, that LL negatively correlates with M_{hip} , the moment arm associated with M_{hip} was calculated as the perpendicular distance from the hip joint to the gravitational force vector of COM_{HAT} (Figure 2.1), approximated as the 2-min average A-P distance between COM_{HAT} and the greater trochanter marker in the posture analysis. Associations between the M_{hip} moment arm and LL measurements were tested using linear regression and correlation analysis.

To test **H2**, that LL is predicted by the passive bending of the lumbar spine due to the mass of the trunk, the lumbar spine was modeled as a multi-jointed beam. This approach is often used to predict

simple bending behavior in experimental studies of vertebrate spinal columns (e.g. Gal, 1993; Long et al., 1997). Although each intervertebral joint experiences local bending moments (Vette et al., 2011; Wagner et al., 2012), bending of the lumbar spine as a whole causes small deformations between vertebral elements that become distributed along the column's length, approximating deformations of a continuous and homogenous beam (Etnier, 2001). In this study, the bending moments acting about the lumbar spine were modeled at the midpoint of the beam's curved length (approximated at the spinous process of L3) to estimate overall deflection relative to its radius of curvature. Gravitational forces acting on COM_{HAT} were assumed to generate sustained, external bending moments to flex the spine anteriorly. The relationship between bending moments (M_{lumbar}) and curvature can be described by the following equation (Bucciarelli, 2009):

Equation 2
$$M_{lumbar} = \frac{EI}{r}$$

where E is the lumbar spine's elastic modulus, I is the second moment of area of its cross section, and r the radius of its curvature. Rearranging Equation 1, substituting into Equation 2, and omitting variable E (which was not measured in this study) yields the following proportional relationship:

Equation 3
$$\theta \propto \frac{M_{lumbar} L}{I}$$

where θ represents the degree of curvature of a loaded beam. M_{lumbar} was calculated as the product of gravity, 38.4% of body mass (approximating the mass of the body above the L3 vertebral level; Vette et al., 2011), and the M_{lumbar} moment arm (the mean A-P distance between COM_{HAT} and the L3 spinal marker from the posture analysis). L was measured as the curved length of the lumbar spine from MRI scans (see Equation 1). The second moment of area, I, was approximated using the mean CSA of the L1-L5 vertebral bodies, since the beam's second moment of area should be proportional to the area of the lumbar vertebral column's cross section. Since curvature of the lumbar spine under load was assumed to

behave similarly across lordosis measurements, LL could be substituted for θ in the beam equation. However, in standing posture M_{lumbar} causes forward flexion of the lumbar spine rather than extension (as assumed by EQ. 3), which decreases curvature of the beam. For this reason, the proportionality in Equation 3 was predicted to be inversely proportional to LL in the beam model, yielding the following relationship:

Equation 4
$$\frac{1}{LL} \propto \frac{M_{lumbar} L}{I}$$

Thus, LL was predicted to correlate negatively with the proportionality represented by the right side of Equation 4. Associations between the beam model and LL measurements were tested using linear regression and correlation analysis.

To test **H3**, that the tradeoff between RTS and ROM explains LL variability, muscle CSA was considered a proxy for trunk strength, since a muscle's CSA is proportional to its capacity to generate force (Ikai and Fukunaga, 1968). Relative trunk strength (RTS) was quantified as the ratio of hypaxial to epaxial muscle size in the following ratio:

Equation 5
$$RTS = \frac{RA}{ES + MF + QL}$$

where RA is the rectus abdominus, ES is the erector spinae, MF is the multifidus, and QL is the quadratus lumborum. Although QL is developmentally a hypaxial muscle, it was included with the epaxial muscle group because of its functional similarity to the other extensor muscles of the back. Relative trunk strength and ROM were used as the independent variables in multiple regression models against LL as the dependent variable. A statistical interaction between RTS and ROM was also tested. Controlling for the effects of ROM and RTS in these models, LL was predicted to be negatively associated with RTS and positively associated with ROM. To further explore the nature of the strength-flexibility interaction, a

post-hoc multiple regression analysis was conducted using hypaxial (RA) and epaxial (ES + MF + QL) muscle group CSAs as two independent predictors of ROM as the dependent variable. In these models, back muscle size was predicted to negatively associate with ROM when accounting for abdominal muscle size as a covariate. Additionally, to test the possibility that greater overall strength is associated with lumbar curvature, correlations were tested between LL and each trunk muscle CSA independently, as well as between LL and total trunk musculature (ES + MF + QL + RA). Muscle CSAs were standardized for body size by dividing muscle CSA by the CSA of the inferior endplate of the L4 vertebral body measured from axial MRI scans.

Statistical analyses

Data processing and analyses were performed using R statistical software (version 3.2.1). A Shapiro–Wilk test was used to examine assumptions of normality in the distributions of each variable. Because ROM and RTS were log-normally distributed, the common logarithms of these variables were used in statistical models. Holm-Bonferroni corrected pairwise *t*-tests were used to compare the three LL measurements. Pearson’s correlations were used to test bivariate associations between continuous variables. Rather than set an alpha level, effect sizes and associated *p*-values are reported and discussed below. R-squared values from ordinary least-squares regression models were used to compare the proportion of variance explained by the three main hypotheses. To compare effect sizes among models testing **H3**, log-transformed variables were converted to *z*-scores and standardized coefficients (β) are reported for multiple regression effects.

Results

Table 2.1: Mean and standard deviation of measurements.

Anthropometrics	
Age, years	22.8 ± 4.2
Height, m	1.72 ± 0.09
Body Mass, kg	65.6 ± 11.4
Body Mass Index, kg/m ²	22.0 ± 2.5
Lordosis measurements	
Cobb LA, deg	49.4 ± 9.5
Supine CA, deg	51.7 ± 17.5
Standing CA, deg	41.2 ± 17.2
Hip moments	
Hip flexor moment arm, mm	4.7 ± 11.2
Beam model variables	
L3 moment arm, cm	10.4 ± 1.2
Bending moments, N · m	37.1 ± 9.6
Lumbar spine length, cm	18.4 ± 8.7
Avg. L1-L5 vertebral body CSA, mm ²	1093.0 ± 157.0
Trunk muscle cross-sectional areas	
Multifidus (MF), mm ²	1253.5 ± 507.2
Erector spinae (ES), mm ²	2617.9 ± 714.1
Quadratus lumborum (QL), mm ²	1094.9 ± 397.9
Rectus abdominus (RA), mm ²	1213.7 ± 353.6
Lumbar range of motion	
Maximum flexion, deg	192.1 ± 3.3
Maximum extension, deg	130.1 ± 20.1
Range of motion, deg	60.5 ± 15.1

Comparing between LL measurement methods, there was no difference between mean Cobb LA and Supine CA ($p = 0.51$), but Standing CA was 20% lower than Cobb LA ($p = 0.08$) and 26% lower than Supine CA ($p = 0.03$; Table 2.1). Cobb LA was strongly correlated with both Supine CA ($r = 0.67$; $p < 0.0001$) and Standing CA ($r = 0.74$; $p < 0.0001$), but the two central angle methods correlated only moderately with each other ($r = 0.46$; $p < 0.01$). The CA methods also captured more variability than

Cobb LA, which showed standard deviations that were 84% smaller than Supine CA ($p = 0.002$) and 81% smaller than Standing CA ($p = 0.002$).

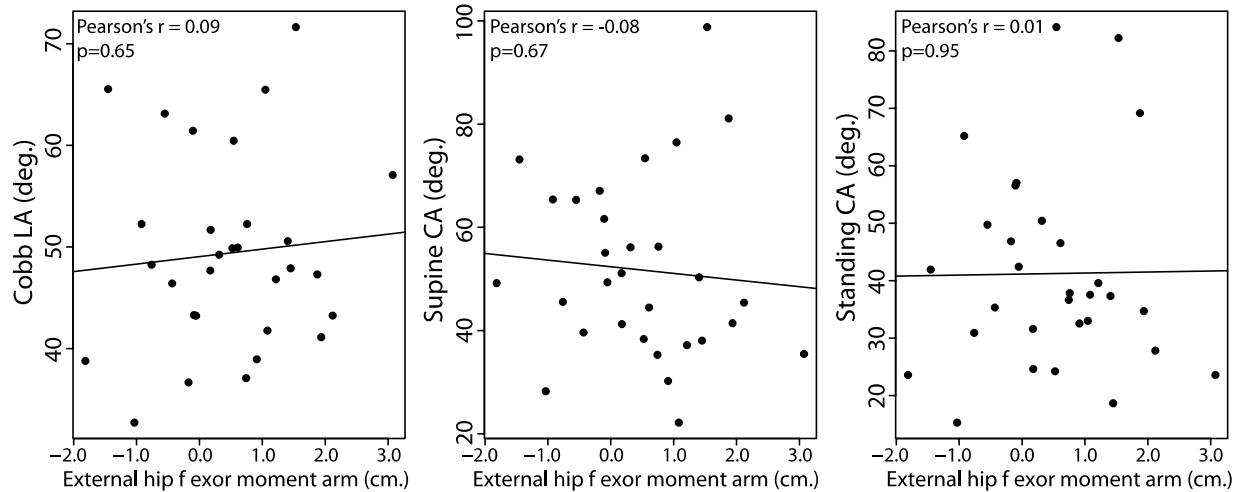


Figure 2.4: Results testing **H1**, that lumbar lordosis is negatively correlated with the external hip flexor moment arm (M_{hip}). No association was found between lordosis and M_{hip} . Positive moment arms indicate that the position of the center of mass of the upper body was anterior to the hip joint, while negative hip moment arms indicate it was posterior to the hip.

Hip moments were not associated with any of the three measures of LL (Figure 2.4). The M_{hip} moment arm showed weak correlations with Cobb LA ($r = 0.09$; $p = 0.65$), Supine CA ($r = -0.08$; $p = 0.67$), and Standing CA ($r = 0.01$; $p = 0.95$). The position of COM_{HAT} was almost directly above the hip joint with a mean position 4.7 ± 11.2 mm anterior to the greater trochanter (Table 2.1). There was also low intra-individual variation in the position of COM_{HAT} during the posture analysis. Average within-trial standard deviation of the M_{hip} moment arm was 1.9 mm over the duration of the 2-min trials.

Table 2.2: Correlations between lordosis and beam model variables

	Pearson's r	P-value
Cobb LA		
Lumbar length (L)	-0.02	0.92
Bending moment (M_{lumbar})	0.25	0.18
Avg. lumbar cross-sectional area (I)	0.12	0.53
Supine CA		
Lumbar length (L)	-0.52	0.003
Bending moment (M_{lumbar})	-0.13	0.49
Avg. lumbar cross-sectional area (I)	-0.1	0.61
Standing CA		
Lumbar length (L)	-0.57	0.001
Bending moment (M_{lumbar})	-0.43	0.02
Avg. lumbar cross-sectional area (I)	-0.33	0.08

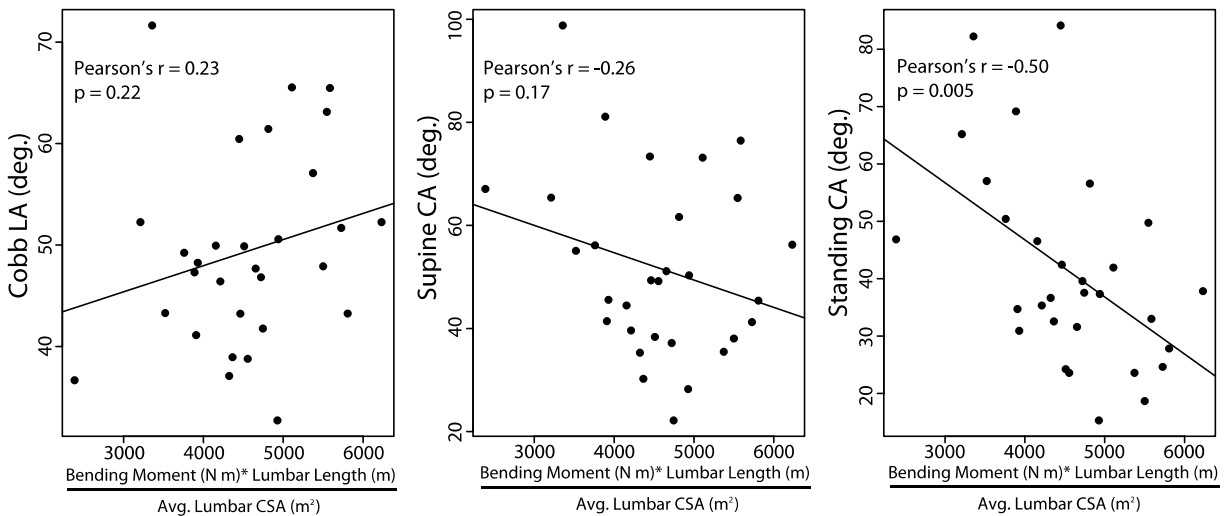


Figure 2.5: Results testing H2, that lordosis is predicted by a beam bending model. The quantity derived in Equation 3 was used as the independent variable in regression models to predict variations in lumbar lordosis (LL). LL measured in the supine position (Cobb LA and Supine CA) were inconsistent in their associations and not strongly correlated with lumbar curvature, but Standing CA showed a strong negative correlation with the beam model.

The beam model predicted by Equation 4 explained 25% of the variation in Standing CA ($r = -0.50$, $p = 0.005$), but the model did not strongly predict Cobb LA ($r = 0.23$, $p = 0.22$) or Supine CA ($r = -0.26$, $p = 0.17$; Figure 2.5). Testing bivariate associations between LL and the beam variables independently, Standing CA was negatively correlated with L ($r = -0.57$, $p = 0.001$) and M_{lumbar} ($r = -0.43$, $p = 0.02$), but only moderately negatively correlated with lumbar body CSA at levels approaching conventional significance ($r = -0.33$; $p = 0.08$; Table 2.2). There was also a strong negative correlation between Supine CA and L ($r = -0.52$, $p = 0.003$).

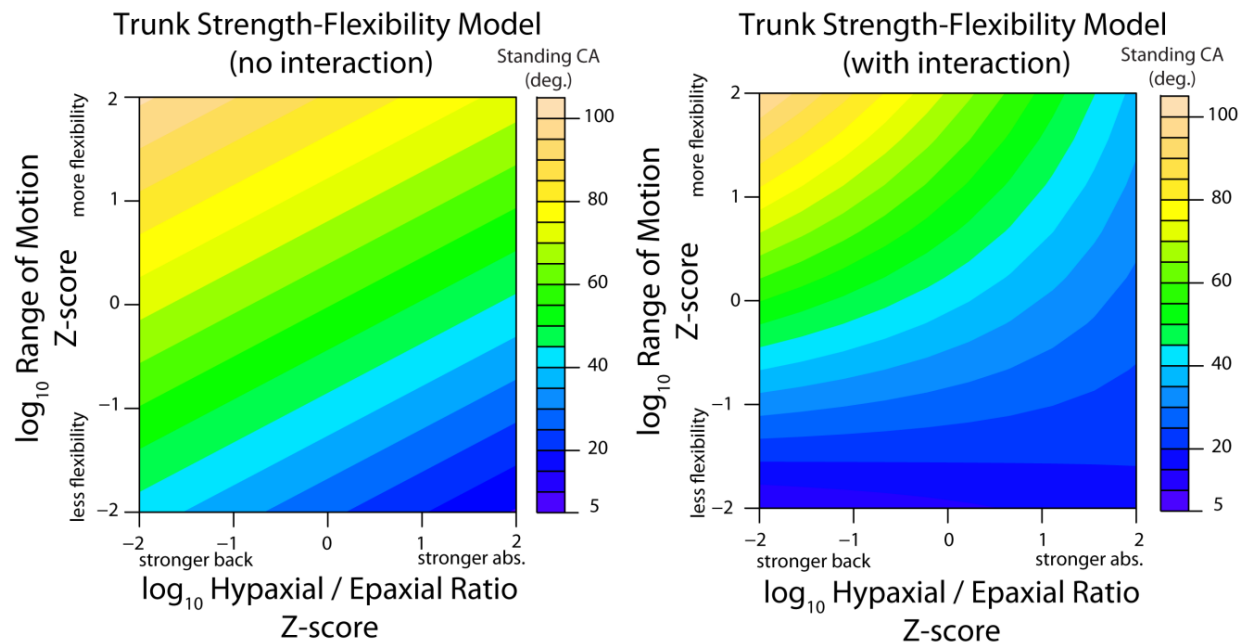


Figure 2.6: A heat map illustration of the strength-flexibility model (**H3**) shown with (right) and without (left) the inclusion of a statistical interaction between lumbar range of motion (ROM) and relative trunk strength (RTS) ratio of hypaxial (abdominal) vs. epaxial (back) muscle size. The figure was generated using coefficient estimates from the multiple regression model of Standing CA predicted by ROM and RTS (Table 2.3), presented here as Z-scores. Higher lumbar curvature was associated with increased lumbar flexibility and relatively stronger back muscles, while straighter lumbar spines were associated with reduced lumbar flexibility and relatively stronger abdominal muscles. The interaction model suggests that variation in lordosis is most strongly affected by RTS among individuals with high ROM, but RTS has little effect on individuals with limited lumbar ROM.

The strength-flexibility model best predicted variations in LL (Table 2.3, Figure 2.6). The interaction model explained 65% of variation in Standing CA ($p = 0.00001$) and 46% of variation in Supine CA ($p = 0.002$), but only 18% of variation in Cobb LA ($p = 0.07$). In all strength-flexibility models tested, RTS had a negative association with LL ($-0.43 < \beta < -0.34$) indicating that a 12% relative increase in abdominal muscle size was associated with a 6.7° decrease in Standing CA. Lumbar flexibility was also consistently and positively associated with LL in all models ($0.23 < \beta < 0.81$), with a 6% increase in ROM associated with a 14.0° increase in Standing CA. There was also a small-to-moderate interaction effect ($-0.27 < \beta < -0.13$) between RTS and ROM. This interaction (Figure 2.6) indicates that RTS had a large effect on LL among individuals with increased ROM. However, among those with decreased ROM, RTS had a small effect on LL. Those individuals tended to have low degrees of lumbar curvature regardless of variation in trunk strength. When included as a covariate, sex had no effect on the strength-flexibility interaction model ($p = 0.58$) (Appendix Table 1.1). The post-hoc multiple regression, which tested the independent effects of hypaxial and epaxial muscle size as predictors of ROM (Figure 2.7), showed that there was no association between ROM and abdominal muscle size ($\beta = 0.41$, $p = 0.16$) but a strong negative association between ROM and back muscle size ($\beta = -0.81$, $p = 0.009$). Furthermore, no bivariate associations were found between LL and size-standardized measurements of any individual trunk muscle CSA ($p > 0.26$), with the exception of a moderate correlation between relative QL size and Cobb LA ($r = 0.38$, $p = 0.04$; Table 2.4). The size-standardized sum of muscle CSAs, measuring total trunk musculature, showed no associations with any measurement of LL ($p > 0.28$).

Table 2.3: Results of the strength-flexibility model regressions

Cobb LA	Main effects model			Interaction model		
	β	S.E.	P-value	β	S.E.	P-value
Intercept	-0.07	0.17	0.64	-0.03	0.17	0.86
log ₁₀ RTS	-0.35	0.18	0.06	-0.38	0.18	0.05
log ₁₀ ROM	0.23	0.18	0.21	0.28	0.19	0.15
log ₁₀ RTS : log ₁₀ ROM				-0.13	0.14	0.36
Multiple R ² = 0.15			Multiple R ² = 0.18			
Overall P value = 0.14			Overall P value = 0.07			
Supine CA	β	S.E.	P-value	β	S.E.	P-value
Intercept	-0.08	0.14	0.60	0.01	0.14	0.95
log ₁₀ RTS	-0.37	0.15	0.02	-0.43	0.14	0.01
log ₁₀ ROM	0.48	0.15	0.005	0.59	0.15	0.001
log ₁₀ RTS : log ₁₀ ROM				-0.27	0.11	0.02
Multiple R ² = 0.32			Multiple R ² = 0.46			
Overall P value = 0.008			Overall P value = 0.002			
Standing CA	β	S.E.	P-value	β	S.E.	P-value
Intercept	-0.06	0.12	0.65	0.02	0.11	0.84
log ₁₀ RTS	-0.34	0.13	0.02	-0.39	0.12	0.003
log ₁₀ ROM	0.7	0.13	0.00002	0.81	0.12	0.000001
log ₁₀ RTS : log ₁₀ ROM				-0.26	0.09	0.01
Multiple R ² = 0.54			Multiple R ² = 0.65			
Overall P value = 0.00006			Overall P value = 0.00001			

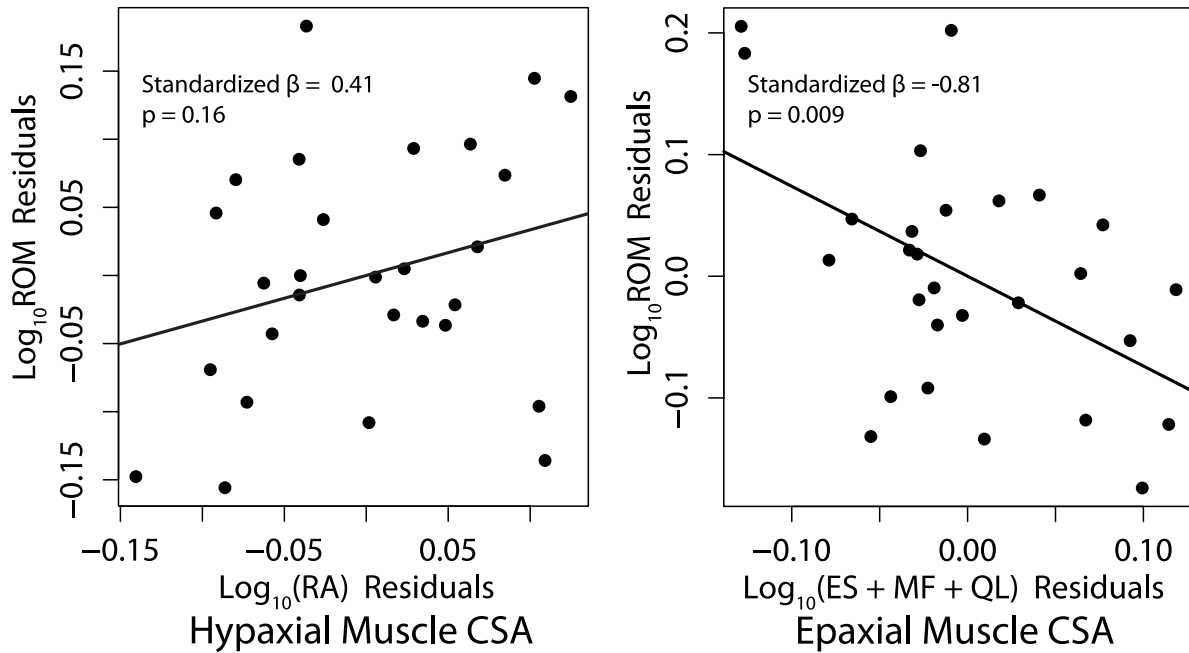


Figure 2.7: Partial regression plots showing the effects of a post-hoc multiple regression analysis of hypaxial (abdominal) and epaxial (back) muscle CSA as separate predictors of lumbar range of motion (ROM). Although ROM shows a slight positive association with abdominal muscle strength, there is a strong negative association between ROM and back musculature, which indicates that the tradeoff between trunk muscle strength and lumbar flexibility may be more affected by the epaxial muscles than the hypaxial muscles.

Table 2.4: Correlations between lordosis and trunk muscle size

Size-Standardized						
Muscle CSA	Cobb LA		Supine CA		Standing CA	
	Pearson's r	P-value	Pearson's r	P-value	Pearson's r	P-value
Erector Spinae (ES)	0.2	0.28	0.02	0.91	-0.15	0.42
Multifidus (MF)	-0.08	0.67	-0.14	0.46	-0.14	0.47
Quadratus Lumborum (QL)	0.38	0.04	0.19	0.3	-0.09	0.65
Rectus Abdominus (RA)	0.003	0.99	-0.12	0.53	-0.22	0.26
Total Musculature (ES + MF + QL + RA)	0.18	0.33	-0.02	0.94	-0.21	0.28

Discussion

The purpose of this study was to test three non-mutually exclusive biomechanical models to explain inter-individual variations in human lumbar lordosis. No support was found for **H1**, that lumbar curvature is associated with moments generated by the mass of the upper body relative to the hip. Hip moments were found to be uncorrelated with LL because COM_{HAT} was consistently positioned almost directly above the hip joint. Since greater LL is often accompanied by increased A-P pelvic tilt (Walker et al., 1987; Vialle et al., 2005; Bailey et al., 2016), it is likely that rotation of the pelvis compensates for lordotic variations, maintaining the orientation of the trunk relative to the acetabulum and reducing hip torque regardless of LL. Since sagittal hip moments appear to be constrained, the results of this study may provide support for the hypothesis that adaptations for bipedal trunk posture have been under strong, possibly stabilizing selection pressures through hominin evolution (Lovejoy, 1988, 2005; Lovejoy and McCollum, 2010). These results are also consistent with previous studies showing that pregnant women increase LL to compensate for increased fetal mass while maintaining a relatively constant vertical orientation of COM_{HAT} over the hip joint throughout pregnancy (Whitcome et al, 2007).

Bending of the lumbar spine in flexion due to the mass of the trunk (**H2**) was found to explain 25% of the variation in standing but not supine LL. The stronger association between Standing CA and the beam model prediction is likely explained by posture: COM_{HAT} does not generate anterior flexion moments to load the lumbar spine in a supine position. Another key finding of the beam model is that longer lumbar spines, higher A-P bending moments, and larger lumbar vertebral bodies were individually associated with decreased standing lordosis. These associations may shed light on potential causes of LL variations related to upper body size, shape, and proportions. For example, predictions from the beam model could explain why Neanderthals had reduced degrees of lordosis compared to other hominins (Been et al., 2012). With deep rib cages and longer pubic bones (Rak and Arensburg,

1987; Franciscus and Churchill, 2002), Neanderthals may have had antero-posteriorly expanded upper bodies and a relatively anteriorly positioned COM_{HAT} , thereby passively increasing lumbar bending moments and perhaps partially explaining their straighter lumbar spines.

The results of this study most strongly support the strength-flexibility model (**H3**), which explained considerably more variation than the other models tested. Although the association between LL and abdominal versus back muscle function has been reported previously (Kim et al., 2005), our study is unique in demonstrating how lumbar mobility differentially interacts with trunk strength to affect lordotic curvature. To explore the nature of this interaction, the post-hoc multiple regression of epaxial and hypaxial muscle size as independent predictors of lumbar mobility revealed that ROM was negatively associated with back muscle size when controlling for abdominal muscle CSA as a covariate, suggesting a tradeoff between back musculature and lumbar mobility. We also tested the possibility that greater overall physical strength would show correlated changes in individual or overall trunk muscle size that could result in differences in LL. However, no strong correlations were found between individual muscle size and any measure of LL with the exception of a moderate correlation between QL and Cobb LA. These results suggest that people who have stronger trunk muscles show no differences in LL compared to individuals with less. The lack of association between muscle CSA and LL is perhaps explained by the function of the back and abdominal muscles in opposition to one another. As Been and Kalichman (2014) note, it is likely that the equilibrium between trunk flexors and extensors, rather than the strength of any individual muscle group, drives variations in lordosis. Indeed, previous work has shown that trunk flexors and extensors have mechanistically linked effects on lumbar curvature (Wagner et al., 2012). Thus, analyzing the ratio of abdominal versus back function strongly predicts lordosis (Kim et al., 2005), though associations between LL and individual muscles may not reveal strong associations.

While greater muscle strength typically increases joint stiffness, the mechanism underlying the relationship between muscle strength and lumbar flexibility is not fully apparent. Research has shown that muscle hypertrophy can result in changes to overall muscle length via an increased number of sarcomeres in series or decreased sarcomere length depending on whether the muscle contracts eccentrically or concentrically (Schoenfeld, 2010). Cross-sectional muscular hypertrophy also may be constrained by the helical structure of surrounding fascial tissue (Scarr, 2016), leading to a reduction in resting muscle length and increased passive stiffness relative to the opposing hypaxial or epaxial trunk muscle group. This constraint could cause A-P imbalance in the tension contributions of abdominal versus back muscles, yielding spinal curvature in the direction of the stronger muscle group while limiting mobility. However, future studies are necessary to fully understand the underlying causes of the strength-flexibility tradeoff.

These findings have important clinical as well as evolutionary implications. From an evolutionary perspective, the strength-flexibility interaction model could be taken to imply that Neanderthals and other hominins with low degrees of LL had less compliant lumbar spines with reduced sagittal ROM regardless of trunk strength variations. However, whether the straighter and possibly stiffer lower backs of Neanderthals had a functional impact remains a matter of conjecture. Since reduced lumbar curvature tends to be associated with lower amounts of intervertebral shearing forces during *in vivo* static loading experiments (Meakin et al., 2008), this may be evidence that Neanderthal spines were selected for increased spinal stiffness and stability, perhaps as an adaptation for supporting substantial loads. These loads could have been the result of behaviors such as carrying heavy objects or rigorous upper body use (Been et al., 2012). However, Neanderthal lumbar spines also supported a greater upper body volume compared to modern humans. In addition to A-P expanded dimensions of the thorax and pelvis, the Neanderthal trunk was mediolaterally broad (Sawyer and Maley, 2005; Gomez-Olivencia et al., 2009; Garcia-Martinez, 2014), resulting in an overall larger upper body compared to modern

humans. The expanded size of the Neanderthal upper body would have generated heavier anatomical loads on the lumbar spine (Equation 4), which would have been stably supported by a straighter vertebral column. We therefore speculate that the Neanderthal lumbar spine, which is characterized by low degrees of LL and possibly reduced lumbar flexibility and greater spinal stability, may have been adapted to resist either extrinsic behavioral loads (e.g. carrying heavy objects) or heavier anatomical loads due to expansion of upper body dimensions. However, the effects of overall expansion in trunk geometry are likely to be the stronger selective pressure due to the sustained passive loading of the spine.

From a clinical perspective, several studies have demonstrated an association between LL and lumbar pathology (Berlemann et al., 1999; Umehara et al., 2000; Kumar et al., 2001; Chen et al., 2009). Because some tissue properties are plastic and can be modified via exercise and other physical therapies, the strength-flexibility model may provide information about effective methods for adjusting lumbar spinal alignment. If changes in RTS and ROM modify LL within an individual, this study suggests that exercises to strengthen trunk muscles are more likely to be effective at modifying spinal posture in individuals with naturally flexible lumbar spines, or when muscle strengthening is accompanied by stretching to increase lumbar range of motion. Strengthening the trunk muscles to modulate lumbar posture is unlikely to be effective in individuals with naturally low lumbar flexibility and in people with immobile lumbar spines, such as those with lumbar spinal fusion. In addition, preservation of normal spinal mobility and muscle activation are crucial for dynamic spinal function. Since abdominal and back muscles are cyclically activated during locomotion, the strength-flexibility model provides a framework for understanding lumbar stability and postural modulation in response to variations in loading patterns.

Several methodological features of this study are also clinically relevant. Posture studies often assess spinal curves in relation to a plumb line or the body's line of gravity (e.g. Schwab et al., 2006), but

these techniques can lead to imprecision when considering the loading forces acting on the lumbar spine. For example, the position and mass of the lower limbs may affect the body's line of gravity in a standing posture, but body segment masses that are below the vertebral column do not load the spine to impact its shape. Therefore, the models tested in this study focused only on the biomechanics of the upper body. Furthermore, one important goal of this study was to compare between different methods for measuring lumbar curvature. Many techniques exist for quantifying LL, but we chose to measure LL using Cobb LA because it has been shown to be a simple and reliable radiological measurement used widely across posture studies (Vrtovec et al., 2009). Nonetheless, the simplicity of the Cobb method (measuring the intersection of vertebral endplate lines) may limit its ability to quantify a wide range of variability in LL (Been and Kalichman, 2014). We therefore compared the Cobb method to the Central Angle method, which quantifies lordosis as the ratio of the lumbar spine's arc length divided by its radius of curvature. Results showed that Cobb LA strongly correlates with both Supine and Standing CA, but the Cobb method captures only a small proportion of variation in LL compared to the CA method. The CA method also outperformed the Cobb method in the biomechanical models tested, showing stronger associations than Cobb LA in models **H2** and **H3**. These findings suggest that Cobb LA is a poor predictor of lordosis, and the Central Angle method offers a better alternative, capturing more variation than Cobb LA while providing a size-standardized measurement of spinal curvature that can be directly implemented in biomechanical studies of the vertebral column.

Study limitations

This study has several limitations. First, the study relies on a sample of healthy young adults (mostly students), sampling less variability than exists among adults from different ages and backgrounds. Nonetheless, the study sampled a wide range of body sizes and proportions (Table 2.1), mitigating some of these potential biases. Second, because we had no access to a standing MRI, this

study relied on skin surface techniques for measuring standing LL. Though we demonstrate that the Standing CA method is correlated with conventional supine MRI measurements (i.e. Cobb LA), it is likely less accurate than standing radiographic imaging due to variations in subcutaneous tissue thickness and vertebral spinous process length. Third, the beam model of **H2** relies on the assumption that the multi-jointed human spine behaves like a homogenous beam-like unit. While more complex models with greater resolution of intervertebral joint motion and bending moments have been developed (Wagner et al., 2012; Vette et al., 2011), the multi-jointed beam method has been shown to provide reliable estimates of overall lumbar deformation in other species ((e.g. Gal, 1993; Long et al., 1997; Etnier, 2001) and therefore offers a novel comparative approach for studying human spinal loading. Furthermore, the assumption that the lumbar spine can be modeled as a homogenous beam may be justified because mean trunk density for males and females is relatively uniform in the lumbar region and differs by only 0.1 g/cm^3 along the trunk from L1 through L5 (Wicke et al., 2008). A related limitation of the beam model is that the elastic modulus of the lumbar spine was not measured, and the second moment of area of the beam was assumed to be proportional to the average CSA of lumbar vertebral bodies. The future inclusion of improved measures of variables E and I in the beam equations may increase the predictive power of this model. Finally, this study used muscle CSA to approximate the functional contributions of trunk muscles to spinal posture, but this method provides only a rough estimate of true physiological muscle recruitment and force production. Other muscles (e.g. psoas, obliques) may affect lumbar curvature as well. Because this study focused on testing a simple, sagittal plane model of the lumbar spine, only the major anterior and posterior intrinsic trunk muscles were included. Future studies might consider including other muscle groups, muscle moment arms, and muscle activation patterns to measure the role of muscle recruitment and function on LL variations.

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References

- Abitbol MM. 1988. Effect of posture and locomotion on energy expenditure. *Am J Phys Anthropol* 77: 191–9.
- Bailey JF, Sparrey CJ, Been E, Kramer PA. 2016. Morphological and postural sexual dimorphism of the lumbar spine facilitates greater lordosis in females. *J Anat* 229(1): 82-91.
- Been E, Gómez-Olivencia A, Kramer PA. 2012. Lumbar lordosis of extinct hominins. *Am J Phys Anthropol* 147: 64–77.
- Berlemann U, Jaszczyszky DJ, Bühler DW, Harms J. 1999. The role of lumbar lordosis, vertebral end-plate inclination, disc height, and facet orientation in degenerative spondylolisthesis. *J Spinal Disord* 12: 68-73.
- Bonmatí A, Gómez-Olivencia A, Arsuaga JL, Carretero JM, Gracia A, Martínez I, Lorenzo C, de Castro JM, Carbonell E. 2010. Middle Pleistocene lower back and pelvis from an aged human individual from the Sima de los Huesos site, Spain. *Proc Natl Acad Sci USA* 107(43): 18386-91.
- Botsford DJ, Esses SI, Ogilvie-Harris DJ. 1994. In vivo diurnal variation in intervertebral disc volume and morphology. *Spine* 19: 935-40.
- Bramble DM, Lieberman DE. 2004. Endurance running and the evolution of Homo. *Nature* 432(7015): 345-52.
- Bucciarelli LL. 2009. Engineering mechanics for structures. New York: Dover Publications.
- Castillo ER, Lieberman DE. 2015. Lower back pain. *Evol Med Public Health*. 1: 2-3.
- Chen IR, Wei TS. 2009. Disc height and lumbar index as independent predictors of degenerative spondylolisthesis in middle-aged women with low back pain. *Spine* 34: 1402–9.
- Damavandi M. 2008. Effect of modeling methods on the body and head-neck-trunk moments of inertia calculations in individuals of different morphology. (Unpublished doctoral dissertation or master's thesis). University of Montreal, Canada.
- De Leva P. 1996. Adjustments to Zatsiorsky-Seluyanov's segment inertia parameters. *J Biomech* 29(9): 1223-30.
- Dempster WT. 1955. Space requirements of the seated operator: geometrical, kinematic, and mechanical aspects of the body with special reference to the limbs. WADC Technical Report (Wright-Patterson Air Force Base). 55-159.
- Dumas GA, Poulin MJ, Roy B, Gagnon M, Jonanovic M. 1991. Orientation and moment arms of some trunk muscles. *Spine* 16: 293-303.
- Etnier SA. 2001. Flexural and torsional stiffness in multi-jointed biological beams. *Biol Bull* 200: 1-8.
- Franciscus RG, Churchill SE. 2002. The costal skeleton of Shanidar 3 and a reappraisal of Neandertal thoracic morphology. *J Hum Evol* 42(3): 303-56.

- Ikai M, Fukunaga T. 1968. Calculation of muscle strength per unit cross-sectional area of human muscle by means of ultrasonic measurement. *Int Z Angew Physiol* 26(1):26-32.
- Gál J. 1993. Mammalian spinal biomechanics. I. Static and dynamic mechanical properties of intact intervertebral joints. *J Exp Biol* 174: 247–80.
- García-Martínez D, Barash A, Recheis W, Utrilla C, Torres Sanchez I, Garcia Rio F, Bastir M. 2014. On the chest size of Kebara 2. *J Hum Evol* 70: 69-72.
- Gomez-Olivencia A, Eaves-Johnson KL, Franciscus RG, Carretero JM, Arsuaga JL. 2009. Kebara 2: new insights regarding the most complete Neandertal thorax. *J Hum Evol* 57: 75–90.
- Hanavan EP. 1964. A mathematical model of the human body. AMRL-TR-64-102. AMRL TR. 1–149.
- Kim MS, Chung SW, Hwang C, Lee CK, Chang BS. 2005. A radiographic analysis of sagittal spinal alignment for the standardization of standing lateral position. *J Korean Orthop Assoc* 40: 861–7.
- Kumar MN, Baklanov A, Chopin D. 2001. Correlation between sagittal plane changes and adjacent segment degeneration following lumbar spine fusion. *Eur Spine J* 10: 314-19.
- Lampl M. 1992. Further observations on diurnal variation in standing height. *Ann Hum Biol* 19: 87–90.
- Long JH, Pabst DA, Shepherd WR, McLellan, WA. 1997. Locomotor design of dolphin vertebral columns: bending mechanics and morphology of *Delphinus delphis*. *J Exp Biol* 200: 65–81.
- Lovejoy CO, McCollum MA. 2010. Spinopelvic pathways to bipedality: why no hominids ever relied on a bent-hip-bent-knee gait. *Philos Trans R Soc Lond B Biol Sci* 365: 3289–99.
- Lovejoy CO. 1988. Evolution of human walking. *Sci Am* 259: 118–25.
- Lovejoy, CO. 2005. The natural history of human gait and posture: Part 1. Spine and pelvis. *Gait Posture* 21(1): 95-112.
- Macintosh JE, Bogduk N. 1991. The attachments of the lumbar erector spinae. *Spine* 16: 783–92.
- Macintosh JE, Bogduk N. 1986. The biomechanics of the lumbar multifidus. *Clin Biomech* 1: 205-13.
- McGill SM, Santaguida L, Stevens J. 1993. Measurement of the trunk musculature from T5 to L5 using MRI scans of 15 young males corrected for muscle fibre orientation. *Clin Biomech* 8: 171-8.
- Meakin JR, Hukins DW, Aspden RM. 1996. Euler buckling as a model for the curvature and flexion of the human lumbar spine. *Proc Biol Sci* 263: 1383–7.
- Meakin JR, Smith FW, Gilbert FJ, Aspden RM. 2008. The effect of axial load on the sagittal plane curvature of the upright human spine in vivo. *J Biomech* 41: 2850–4.
- Pal GP, Routal RV. 1999. Mechanism of change in the orientation of the articular process of the zygapophyseal joint at the thoracolumbar junction. *J Anat* 195(02): 199-209.
- Pilbeam D. 2004. The anthropoid postcranial axial skeleton: Comments on development, variation, and evolution. *J Exp Zool B Mol Dev Evol* 302B(3): 241-67.

- Rak Y, Arensburg B. 1987. Kebara 2 Neanderthal pelvis: first look at a complete inlet. 1987. *Am J Phys Anthropol* 73(2): 227-31.
- Rao G, Amarantini D, Berton E, Favier D. 2006. Influence of body segments' parameters estimation models on inverse dynamics solutions during gait. *J Biomech* 39(8): 1531-6.
- Russo GA. 2010. Prezygapophyseal articular facet shape in the catarrhine thoracolumbar vertebral column. *Am J Phys Anthropol* 142(4): 600-12.
- Saha D, Gard S, Fatone S, Ondra S. 2007. The effect of trunk-flexed postures on balance and metabolic energy expenditure during standing. *Spine* 32: 1605–11.
- Saha D, Gard S, Fatone S. 2008. The effect of trunk flexion on able-bodied gait. *Gait Posture* 27: 653–60.
- Sawyer GJ, Maley B. 2005. Neanderthal reconstructed. *Anat Rec* 283B: 23-31.
- Scarr G. 2016. Fascial hierarchies and the relevance of crossed-helical arrangements of collagen to changes in the shape of muscles. *J Bodyw Mov Ther* 20(2): 377-87.
- Schoenfeld BJ. 2010. The mechanisms of muscle hypertrophy and their application to resistance training. *J Strength Cond Res* 24(10): 2857-72.
- Schwab F, Lafage V, Boyce R, Skalli W, Farcy JP. 2006. Gravity Line Analysis in Adult Volunteers. *Spine* 31: E959-67.
- Shapiro LJ. 1993. Functional morphology of the vertebral column in primates. In: Gebo DL, editor. *Postcranial adaptation in nonhuman primates*. DeKalb (IL): Northern Illinois University Press. p. 121–49.
- Steindler A. 1997. *Kinesiology of the human body under normal and pathological conditions*. Springfield: Charles C Thomas Publisher.
- Strickland AL, Shearin RB. 1972. Diurnal height variation in children. *J Pediatr* 80: 1023-5.
- Thompson NE, Demes B, O'Neill MC, Holowka NB, Larson SG. 2015. Surprising trunk rotational capabilities in chimpanzees and implications for bipedal walking proficiency in early hominins. *Nat Commun* 6: 8416.
- Tillmann V, Clayton PE. 2001. Diurnal variation in height and the reliability of height measurements using stretched and unstretched techniques in the evaluation of short-term growth. *Ann Hum Biol* 28: 195–206.
- Umehara S, Zindrick MR, Patwardhan AG, Havey RM, Vrbos LA, Knight GW, Miyano S, Kirincic M, Kaneda K, Lorenz MA. 2000. The biomechanical effect of postoperative hypolordosis in instrumented lumbar fusion on instrumented and adjacent spinal segments. *Spine* 25: 1617–24.
- Vette AH, Yoshida T, Thrasher TA, Masani K, Popovic MR. 2011. A complete, non-lumped, and verifiable set of upper body segment parameters for three-dimensional dynamic modeling. *Med Eng Phys* 33(1): 70-9.

- Vialle R, Levassor N, Rillardon L, Templier A, Skalli W, Guigui P. 2005. Radiographic Analysis of the sagittal alignment and balance of the spine in asymptomatic subjects. *J Bone Joint Surg Am* 87: 260–7.
- Voss LD, Bailey BJ. 1997. Diurnal variation in stature: is stretching the answer? *Arch Dis Child*. 77: 319–22.
- Vrtovec T, Pernuš F, Likar B. 2009. A review of methods for quantitative evaluation of spinal curvature. *Eur Spine J* 18: 593-607.
- Wagner H, Liebetrau A, Schinowski D, Wulf T, de Lussanet MH. 2012. Spinal lordosis optimizes the requirements for a stable erect posture. *Theor Biol Med Model* 9(1): 13.
- Wainwright SA. 1982. Mechanical design in organisms. Princeton: Princeton University Press.
- Walker ML, Rothstein JM, Finucane SD, Lamb RL. 1987. Relationships between lumbar lordosis, pelvic tilt, and abdominal muscle performance. *Phys Ther* 67: 512–6.
- Ward CV, Latimer B. 2005. Human evolution and the development of spondylolysis. *Spine* 30(16): 1808-14.
- Whitcome KK. 2012. Functional implications of variation in lumbar vertebral count among hominins. *J Hum Evol* 62: 486–97.
- Whitcome KK, Shapiro LJ, Lieberman DE. 2007. Fetal load and the evolution of lumbar lordosis in bipedal hominins. *Nature* 450: 1075–8.
- White A, Panjabi M. 1978. Clinical biomechanics of the spine. Philadelphia: Lippincott.
- Whitehouse RH, Tanner JM, Healy MJ. 1974. Diurnal variation in stature and sitting height in 12-14-year-old boys. *Ann Hum Biol* 1: 103–6.
- Wicke J, Dumas GA, Costigan PA. 2008. Trunk density profile estimates from dual X-ray absorptiometry. *J Biomech* 41(4): 861-7.
- Williams SA. 2012. Placement of the diaphragmatic vertebra in catarrhines: implications for the evolution of dorsostability in hominoids and bipedalism in hominins. *Am J Phys Anthropol* 148(1): 111-22.
- Williams SA, Middleton ER, Villamil CI, Shattuck MR. 2016. Vertebral numbers and human evolution. *Am J Phys Anthropol* 159(S61): S19-S36.
- Willner S. 1981. Spinal pantograph - a non-invasive technique for describing kyphosis and lordosis in the thoraco-lumbar spine. *Acta Orthop* 52: 525–9.

Chapter 3 – Effects of lordosis variability on bending deformations in the human lumbar spine during *in vivo* axial loading

Introduction

Lumbar lordosis, the anterior convex curvature of the lower spine, is an important structural feature of the human vertebral column that facilitates upright posture but varies substantially within modern humans and among fossil hominin taxa (Been et al., 2012). Interpreting the adaptive significance of spinal curvature variations is a challenge because lordosis serves multiple functions. The primary function of lordosis is to balance the mass of the upper body over the hip joint, providing a stable orientation of the trunk in order to cope with the energetic and biomechanical demands of bipedalism (Abitbol, 1988; Saha et al., 2007; Whitcome et al., 2007; Lovejoy and McCollum, 2010). But lordosis also plays a role in modulating the lumbar spine's ability to cope with both internal and external loads. During locomotion, the shape of the lumbar spine influences its response to forces transferred between the upper and lower body, suggesting that dynamic changes in lordosis reflect the lumbar spine's overall viscoelastic properties (Gracovetsky and Iacono, 1987; Syczewaska et al., 1999; Adams et al., 2006). In addition, studies of static axial loading of the vertebral column suggest that lordotic postures may affect the lumbar spine's resistance to deformation (Adams and Hutton, 1985; Patwardhan et al., 1999; Aspden, 1989; Shirazi-Adl and Parnianpour, 1999; Shirazi-Adl et al., 2002; El-Rich et al., 2004; Meakin et al., 2008). However, the relationship between natural variations in lordosis and *in vivo* spinal bending behavior remains unclear.

Despite methodological differences between studies, evidence suggests that lordotic postural variations strongly influence how the spine changes shape and where forces are concentrated during loading. For decades, *ex vivo* experimental studies have examined mechanical properties of individual intervertebral motion segments from cadaveric specimens (e.g. Markolf, 1972; Panjabi et al., 1976; Lin

et al., 1978; Shirazi-Adl et al., 1986; Janevic et al., 1991; Gardner-Morse and Stokes, 2004). Based on analyses of isolated motion segments, researchers have hypothesized that straighter lumbar postures offer advantages including higher compressive strength and reduced stress at the zygapophyses and posterior annulus fibrosus (Adams and Hutton, 1985; Adams et al., 2006). Without the aid of muscles, experimental testing of complete osteoligamentous cadaveric lumbar spines under load reveals large changes in spinal shape, high bending moments, and buckling at very low amounts of force (Crisco, 1989; Patwardhan et al., 1999). Using methods to constrain loading orientation in lordotic postures to mirror physiological loading, however, variations in lordotic postures appear to change the magnitude and pattern of angular deformations between vertebral elements (Patwardhen et al., 1999). Thus, *ex vivo* studies have limited ability to reproduce physiologically realistic loading patterns.

Complementary approaches are to conduct *in vivo* lumbar loading experiments or to use mathematical models to predict loading patterns. *In vivo* studies have shown that individuals tend to reduce lumbar curvature in forward flexion when supporting axial loads (El-Rich et al., 2004; Meakin et al., 2008; Rodriguez-Soto et al., 2013). An explanation for this pattern of spinal deformation may be that straighter spines offer greater stability as predicted by analyses of isolated intervertebral joints (Adams and Hutton, 1985). Furthermore, simple mathematical models of the lumbar spine as an arch support the hypothesis that reduced curvature increases the spine's structural stability (Aspden, 1989), a finding predicted from Euler buckling models of spinal curvature as well (Scholten et al., 1988). More complex finite element models also suggest that straighter lumbar postures have the advantages of decreasing maximum strain in the discs, reducing intervertebral shearing forces and bending moments, and limiting the amount of muscular activity required to stabilize the spine during loading (Shirazi-Adl and Parnianpour, 1999; Shirazi-Adl et al., 2002). However, one *in vivo* study by Meakin et al. (2008) found a puzzling relationship between variations in unloaded lordotic curvature and lumbar deformation: although average lordosis decreased during loading, lumbar spines with naturally low degrees of

curvature tended to straighten while those with high curvature tended to become more curved. This discovery has not been reported in other *in vivo* studies, raising questions about the relationship between lumbar posture and patterns of spinal deformation.

If straighter spines are more stable, then how do they achieve this stability? Do natural variations in spinal shape provide differences in bending resistance allowing straighter spines greater stability? To our knowledge, no studies have quantified the relationship between *in vivo* lumbar bending behavior and underlying natural variations in lordotic posture, so physiologically based estimates of lumbar bending stiffness remain poorly defined. Furthermore, viscoelastic creep response has been well documented in isolated spinal segments (e.g. Adams and Dolan, 1996), but little is known about time-dependent *in vivo* spinal deformation during loading. Do spines tend to straighten over time to maintain stability as suggested by modeling studies, or is the directional pattern of deformation differentially affected by underlying natural variations in lordosis (Meakin et al., 2008)?

To investigate these questions, the present study integrated three approaches to explore how lordosis affects *in vivo* bending deformations in the lumbar spine during axial loading. First, a beam model was used to predict the relationship between unloaded lordosis and bending deformations. Second, radiological imaging was used to correct external measurements of lordosis for internal variations in spinal shape. Finally, *in vivo* weight vest experiments tested axially applied loads during seven loading conditions. The lumbar spine's global deformation response was estimated and the relationship between bending stiffness and lumbar lordosis was quantified.

Hypotheses

Three mutually-compatible hypotheses were tested in this study. The first hypothesis (**H1**) is that lumbar spines tend to straighten on average under load in order to increase stability. The second hypothesis (**H2**) is that variations in the initial degree of unloaded lordosis affect time-dependent changes in lordosis during a loading trial. We predict that patterns of loading deformation across trials will mirror changes in lordosis within trials. Assuming straighter spines offer greater stability, **H2** predicts that straighter postures show less variability in shape over time. In addition, **H2** predicts that more curved spines will show a relative increase in curvature during a loading bout, and straighter spines will show a relative decrease in curvature (Meakin et al., 2008). The final hypothesis (**H3**) tests the relationship between lumbar bending stiffness and natural lordotic curvature. Assuming a beam bending model of the lumbar spine, **H3** predicts that the lumbar spine's bending stiffness and elastic modulus will decrease such that individuals with naturally straighter lordosis will show stiffer spines than individuals with naturally more curved lordosis (see *Model* below).

Materials and Methods

Table 3.1: Glossary of variables and abbreviations

CA	centroidal axis of the beam
COM_{HAT}	center of mass of the upper body (head, arms, and trunk)
E	elastic modulus
ε	bending strain
ε_0	elastic modulus in the unloaded condition
d	average antero-posterior diameter of the lumbar vertebral body
F_{HAT}	force of gravity acting the center of mass of the head, arms, and trunk
GLMM	general linear mixed model
I	second moment of inertia of the beam
i	inner surface of the beam, i.e. posterior border of lumbar vertebral bodies
k	bending stiffness coefficient
L	length of the external surface of the lordotic spine from T12 to S1
M	bending moment
m_{body}	mass of the body
m_{load}	mass of the applied load
NA	neutral axis of the beam
r	radius of curvature along the external surface of the lower back
r_{CA}	radius of curvature through the centroidal axis
r_i	radius of curvature along the inner surface of the beam
r_{HAT}	perpendicular distance from the center of rotation of the beam to the F_{HAT} force vector
r_{NA}	radius of curvature through the neutral axis
σ	bending stress
t	time
TD	tissue depth from skin to the posterior border of the lumbar vertebral bodies
θ	lordosis angle (measured using the Central Angle method)
θ_0	natural lordosis angle in the unloaded condition
y_{max}	max distance from neutral bending axis to the inner surface of the beam, measuring max strain in the direction of the center of rotation of the spine in bending

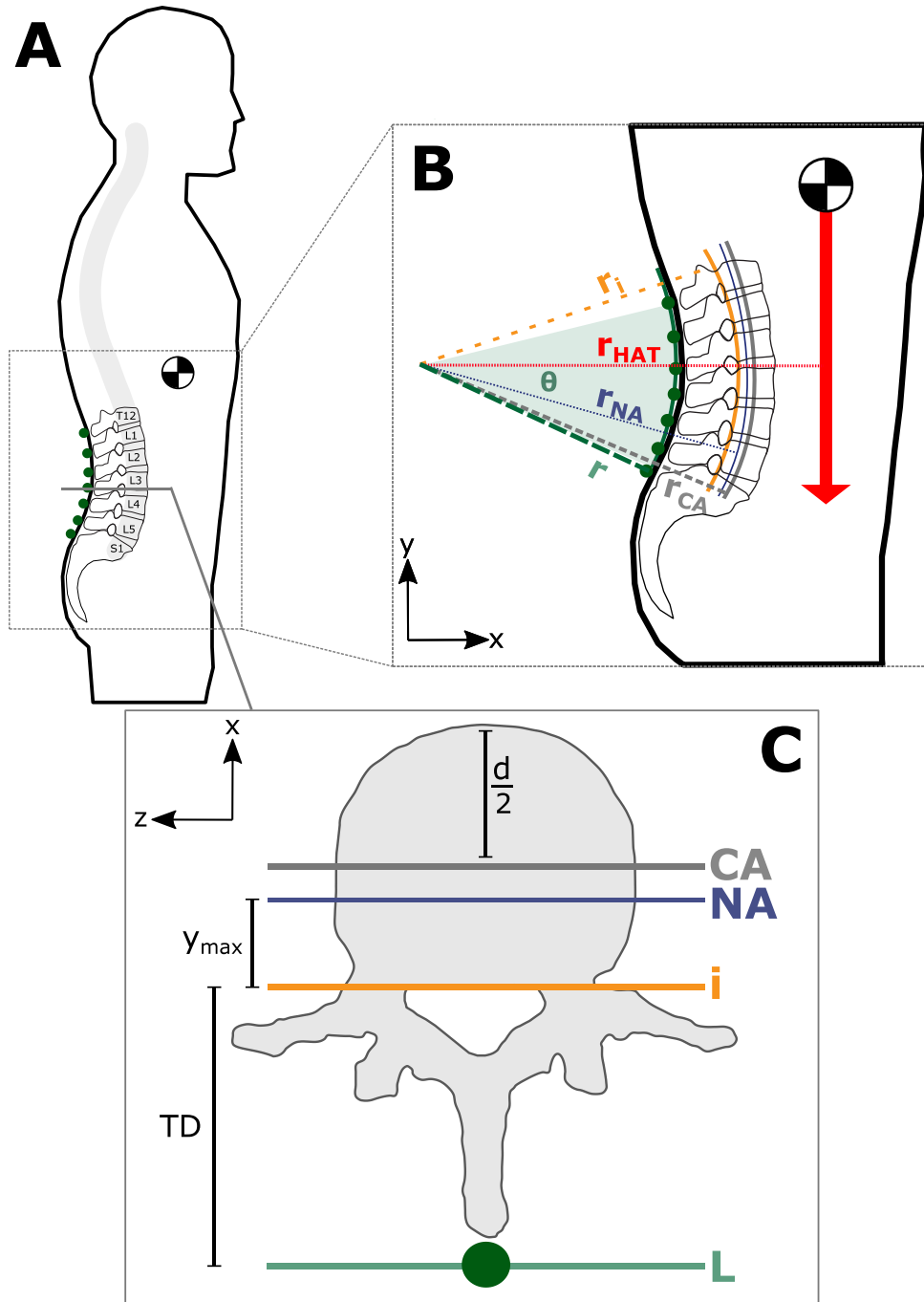


Figure 3.1: (A) Sagittal view of the upper body with its center of mass $\odot$, pre-sacral spine (gray curved line), and outline of the lower spine. External markers (solid green dots) were affixed to the surface of the back from T12 to S1. (B) Beam bending variables. The curved length of the plane along the external surface of the lumbar spine (L, solid green line) divided by its radius of curvature (r , green dotted line) measured lordosis (θ , light green shaded area). Radiological imaging was used to find the radii of curvature to the beam's inner surface (r_i , orange dotted line), centroidal axis (r_{CA} , gray dotted line), and neutral axis (r_{NA} , blue dotted line). The perpendicular moment arm (r_{HAT} , red dotted line) to the force vector acting on the center of mass (solid red line) caused forward flexion bending moments. (C) A coronal slice at the L3 level showing the theoretical locations of planes along the cross section of the beam representing the centroidal axis (CA, gray line), neutral axis (NA, blue line), inner surface (i, orange line), and arc length (L). The distance from NA to i was used to quantify strain at the beam's inner surface (y_{max}). Radiological measurements were used to find tissue depth (TD) and vertebral body diameter (d).

Model

To predict overall bending deformation of the loaded spine, the entire lumbar vertebral column was considered as a single functional spinal unit. The lumbar spine was modeled as a curved flexural member in pure bending to account for the degree of angular deflection in the spine pre-load (Figure 3.1). In contrast to straight beam models, the position of the neutral axis (NA) along the cross section of a curved beam is not the same as the centroidal axis (CA), which differentially affects patterns of stress and strain depending on the direction of the beam's curvature (Dowling 2012). This assumption is supported by empirical evidence that the position of the instantaneous axis of rotation between vertebrae shifts posteriorly as lordotic curvature increases (Patwardhen et al., 1999). Along the plane passing through the points at each intervertebral instantaneous axis of rotation, angular motion between adjacent vertebral bodies is zero, and there is no bending stress. Using modeling equations for curved beams (Dowling, 2012), this uniform shift in the instantaneous axis of rotation is assumed to predict the change in the position of a curved beam's neutral axis.

The curved beam model was bounded superiorly at T12 and inferiorly at S1. The beam was assumed to have homogenous density and a circular, solid cross section. Furthermore, the model assumed an axis of symmetry in a plane along the beam's cross section and that deformations along the length of the beam were uniformly distributed. Despite physiologically relevant variations in the change in orientation of individual vertebral elements under load (Rodriguez-Soto et al., 2013), the net change in overall shape was assumed to be approximated by the total angular deflection of the beam. An additional assumption of the model was that the elastic modulus of tissue elements in tension and compression was equivalent (Dowling, 2012).

The radius of the beam's curvature (r) represents deflection of the spine in lordosis as angle θ such that

Equation 1
$$\theta = \frac{L}{r}$$

where L is the curved length of the lumbar spine measured from the T12 through S1 external markers, and r is the radius of curvature on the external surface of the back (Figure 3.1B). All radii of curvature derived from variable r , representing distances to planes along the cross section of the beam, were assumed to share a common center of rotation. Because internal imaging of the spine under load was not possible in this study, the model relied on external measurements of lordosis, which can provide reliable estimates of loaded spinal shape comparable to internal imaging of the spine under load (e.g. El-Rich et al., 2004). However, external measurements of loaded spinal curvature can be improved when the lengths of the spinous processes are known (Meakin et al., 2008). In order to account for tissue between the external markers and the vertebral bodies, radius measurements were corrected for differences in tissue depth (TD) between the skin's surface and the posterior border of the lumbar vertebral bodies representing the inner surface of the beam (i). Measurements of beam radii were paired with magnetic resonance imaging (MRI) of the lumbar spine to correct for participant-specific TD (see *Imaging* below). As such, the radius of curvature of the inner surface of the beam (r_i) was measured as

Equation 2
$$r_i = r + TD$$

From this value, the position of the centroidal axis (CA) of the beam was computed for the plane passing through the center of the lumbar spine's cross section (Figure 3.1C). The radius of curvature through the CA (r_{CA}) is given by

Equation 3
$$r_{CA} = r_i + \frac{d}{2}$$

where d is the average participant-specific antero-posterior diameter of the T12-S1 lumbar vertebral bodies measured using MRI (see *Imaging* below). The equation for a curved flexural member with a circular solid cross section was then used to find the radius of curvature to the neutral bending axis (r_{NA})(Dowling, 2012):

Equation 4

$$r_{NA} = \frac{d^2}{4(2r_{CA} - \sqrt{4r_{CA}^2 - d^2})}$$

Bending strain at distance y from the beam's NA was calculated as the maximum distance to the inner surface of the beam (y_{max}), corresponding to strain (ϵ) at the furthest plane in the beam in the direction of the axis of rotation. y_{max} was calculated as

Equation 5

$$y_{max} = r_{NA} - r_i$$

Strain was calculated at distance y_{max} along the cross section of the beam, which simplifies to

Equation 6

$$\epsilon = \frac{\Delta L}{L} = \frac{(r_{NA} - y_{max}) \theta - r_{NA} \theta}{r_{NA} \theta} = \frac{-y_{max}}{r_{NA}}$$

Note that by sign convention y_{max} is positive inward, so positive strain represents decreased curvature while negative strain represents increased curvature.

To estimate bending stress (σ), the center of mass of the upper body (i.e. head, arms, and trunk; COM_{HAT}) was modeled as generating bending moments in the direction of forward flexion of the spine. Gravitational forces (F_{HAT}) acting on COM_{HAT} were estimated as

Equation 7

$$F_{HAT} = (0.384 m_{body} + m_{load}) g$$

where m_{body} represents body mass multiplied by 38.4% to estimate the amount of body mass carried above the L3 vertebral level (Vette et al., 2011). This value was added to the mass of the applied

external load (m_{load}), and the resulting sum was multiplied by gravitational acceleration (g) to find the total force acting on the lumbar spine above the center of rotation of the beam (i.e. L3 vertebral level). The moment arm (r_{HAT}) was found as the sum of the antero-posterior distances from the center of rotation of the beam to the external surface of the spine and the distance from the L3 spinal marker to the trunk's gravitational force vector ($r_{L3, HAT}$) such that

Equation 8
$$r_{HAT} = r + r_{L3, HAT}$$

The product of Equations 7 and 8 provided an estimate for total bending moments acting on the lumbar spine (M) as

Equation 9
$$M = F_{HAT} \cdot r_{HAT}$$

Stress σ along the radius of the inner surface of the beam was calculated as

Equation 10
$$\sigma = -\frac{M y_{max}}{I}$$

where I represents the second moment of area of the beam's cross section, estimated for solid circular beams as

Equation 11
$$I = \frac{\pi}{4} \left(\frac{d}{2}\right)^4$$

To calculate the lumbar bending stiffness coefficient (k), the slope of the relationship between changes in M and changes in θ was found by

Equation 12
$$k = \frac{\Delta M}{\Delta \theta}$$

Elastic modulus (E) was similarly calculated as the slope of the relationship between changes in σ and changes in ϵ :

Equation 13

$$E = \frac{\Delta \sigma}{\Delta \varepsilon}$$

Given Equation 13, the predicted relationship between bending resistance and lordosis can be found using an alternative formula for calculating M from variables E, I, and r (Bucciarelli, 2009):

Equation 14

$$M = \frac{EI}{r}$$

Rearranging Equation 1 and substituting for variable r in Equation 14, the following relationship between lordotic curvature and beam bending variables emerges (Castillo et al., 2017):

Equation 15

$$\theta = \frac{ML}{EI}$$

Equation 15 therefore predicts a proportional relationship between θ and E such that

Equation 16

$$\theta \propto \frac{1}{E}$$

Thus, Equation 16 reveals that lumbar lordosis angle θ should be inversely proportional to E (**H3**).

Participants

To test the model, a sample of participants from the greater Boston area was recruited. The study focused on young healthy adults (aged 18-35 years) in order to minimize the potential effects of changes in spinal alignment and pathology associated with age (Schwab et al., 2006). Three different studies over a four-month period were conducted, but only the weight vest experiment is reported here. Twenty-six participants (15 male, 11 female) completed the spinal loading experiment. Participants were recruited to sample a diversity of heights and body masses (Table 3.2). A general health questionnaire was administered prior to participation, and participants were excluded due to any history of back pain, sciatica, scoliosis, major illness, or injury in the three months prior to the study. All

protocols were approved by the Committee on the Use of Human Subjects at Harvard University. Written informed consent was granted prior to participation in the experiments. Study protocols, including MRIs and vest loading experiments, were conducted on different days at the same time in the afternoon between 3-6 PM in order to minimize the potential influence of circadian patterns of the viscoelastic creep in spinal tissues (Strickland and Shearin, 1972; Whitehouse et al., 1974; Lampl, 1992; Botsford et al., 1994; Voss and Bailey, 1997; Tillman and Clayton, 2001).

Table 3.2: Summary of participant anthropometrics

	<u>Mean</u>	<u>SD</u>	<u>Range</u>
Height (m)	1.73	0.09	(1.54, 1.88)
Body Mass (kg)	65.9	11.2	(45.6, 89.5)

Imaging

MRI scans were used to correct external measurements of spinal shape for internal spinal anatomy. A Siemens TIM Trio (3-T) MRI scanner at the Center for Brain Science Neuroimaging Facility of Harvard University imaged the lumbar spine following protocols described in Castillo et al. (2017). In brief, participants were scanned in a neutral, supine position with legs extended and arms resting at their sides using a standard spine-array and large flex coils. A midsagittal, single-slice “localizer” scan (repetition time = 8.6 ms, echo time = 4 ms; 7 mm thickness, 1.7 mm/pixel) was used to orient individuals in the scanner and calculate variables TD and d using ImageJ (National Institutes of Health). TD was quantified as the participant-specific antero-posterior mean distance between the skin’s surface and the posterior borders of T12 through S1 vertebral bodies. The diameter (d) of the lumbar vertebral bodies was measured as the mean antero-posterior diameter of T12-L5 vertebral bodies (Figure 5.1C).

Experiments

Loading experiments were conducted within 45 days of the MRI. To estimate COM_{HAT} , reflective markers were affixed to the following anatomical landmarks: the greater trochanters, anterior and posterior superior iliac spines, iliac crests, acromion processes, medial and lateral humeral epicondyles, ulnar and radial styloid processes, mandibular processes, the sternal notch, and the spinous processes of C7 and T12 through S1.

Similar to weighted apron experiments used by previous studies (Meakin et al., 2008), two weighted vests (TKO model 244WV) were used to apply axial loads to the spine via the shoulders. These flexible, lightweight vests each initially had 38 pockets (16 on the front; 22 on the back) in which loads could be added. The infero-posterior surfaces of vests were altered by cutting a rectangular portion of material measuring approximately 14 x 22 cm, thereby removing six pockets to provide an unobstructed view of the spine and symmetrical anterior and posterior locations for adding loads (16 pockets on both front and back). Each altered vest was capable of applying a maximum of 14.5 kg of added mass using soft, flexible 450-gram weights designed to fit snugly into each pocket. When all pockets were filled to capacity, a second identical vest was draped over the first allowing additional mass to be added up to 30% of an individual participant's body mass (i.e. 27 kg of added mass for the heaviest participant).

At the start of the experiment, participants stood motionless in a comfortable, neutral posture (arms resting comfortably at their sides) for 2 minutes without the vest to measure trunk geometry and the position of COM_{HAT} . The unloaded weight vest was then draped over the shoulders to measure lordosis during the 0% loading trial. Including this trial, a total of seven loading conditions were tested from 0% to 30% body mass in increasing loads of 5% body mass. Because viscoelastic creep occurs in spinal tissues under load (Adams and Dolan, 1996), participants were instructed to stand motionless using a natural posture for 2 minutes for each trial. After two minutes, additional 5% loads were divided to symmetrically apply mass to the front and back of the vest at comparable locations in an attempt to

maintain the approximate position of the unloaded COM_{HAT} . Participants completed all loading trials with the exception of one who was unable to continue with the experiment after the 20% load due to fatigue.

Kinematics

Eight infrared cameras (Oqus 1 Series, Gothenburg, Sweden) and Qualysis tracking software (version 2.10) captured 3-D kinematics at 200 Hz for the 2-min duration of each trial. Since participants were nearly motionless during trials, kinematic data were subsequently filtered using a second-order digital Butterworth filter with a low-pass cutoff of 2 Hz to remove higher frequency noise. Data were then decimated to reduce the sampling rate to a manageable number of frames per trial (4000).

The position of COM_{HAT} was estimated following methods described in Castillo et al. (2017). Briefly, Visual3D (C-Motion, v.5) software estimated the position of COM_{HAT} with body segment masses calculated following Dempster (1955) and inertial properties following Hanavan (1964). Kinematic measurements from the initial vest-free trial were used to estimate COM_{HAT} because key markers on the trunk were obscured by vests during loading trials. Thus, calculating the marker-based position of COM_{HAT} during loading was not possible, and COM_{HAT} was assumed to remain in the same relative position based on the geometry of the trunk. However, the mass of COM_{HAT} varied with the addition of loads, and its position relative to the lumbar spine was modeled based on differences in $r_{L3, HAT}$ according to Equations 7 and 8.

Spinal markers from T12 through S1 were used to quantify lordosis angle θ according to Equation 1. For details and discussion of the reliability of using the Central Angle method, see Castillo et al. (2017). θ was found for each kinematic frame by computing the least-squares, best-fit circle passing through the seven points representing T12-S1. Mean deflection of the beam for each trial was measured

as the 2-min average of angle θ , and the unloaded vest trial was used to measure lordosis at 0% body mass (θ_0).

Hypothesis Testing

The first hypothesis (**H1**) was that lumbar spines show an average tendency to become straighter under load. Mean θ was compared between trials with the prediction that θ positively correlates with applied mass and relative load mass. Since this was a repeated-measures experimental design, this prediction was tested using general linear mixed models (GLMM). The first (Model 1a) tested the association between mean θ and applied load mass, and the second (Model 1b) tested the association between relative changes in lordosis between trials ($\Delta\theta$) and load as a percentage of body mass.

The second hypothesis (**H2**) was that natural variations in lordosis are associated with time-dependent changes in lordosis ($\Delta\theta$) during a loading trial. To test whether straighter spinal postures are more stable during loading, we assumed that lumbar stability was inversely proportional to lordosis variability over time. Variation in θ within a trial was quantified as the standard deviation of θ for each 2-min loading condition. A GLMM (Model 3a) tested for a predicted positive correlation between within-trial standard deviation in θ and mean θ used during that trial. To account for the potential effect of added load, load percentage was included as a model covariate. The second prediction of **H2** was that mean θ affects the direction of $\Delta\theta$ during a loading trial, a pattern reported by Meakin et al. (2008). This prediction was tested by fitting a linear regression to the relationship between θ and time (t) within each trial. The slope $\Delta\theta/\Delta t$ was predicted to be positive among individuals with higher mean θ and negative among individuals with lower mean θ during a trial. Thus, Model 3b tested for a relationship between $\Delta\theta/\Delta t$ as the dependent variable and mean θ as the predictor variable while controlling for load percentage as a covariate.

Finally, **H3** tested the hypothesis that there is an inverse relationship between unloaded lordosis and the lumbar spine's resistance to bending deformations. Between loading trials, the load-displacement and stress-strain curves were found for each individual. Since the linear region of the curves was not known a priori, bending stiffness (k) and elastic modulus (E) were calculated between each successive loading trial, and differences between the slopes at each loading interval were compared using GLMMs to find the linear region. Bending stiffness (k) was found by Equation 12 as the ratio of ΔM to $\Delta \theta$ between successive loading trials. Since forward flexion of the spine was associated with increases in moments, absolute changes in angular deflection were used to calculate stiffness as a positive value. The modulus of elasticity E was similarly found by Equation 13 as the ratio of $\Delta \sigma$ to $\Delta \epsilon$ between successive loading trials. Two GLMMs tested the correlations between k and θ_0 controlling for load percentage (Model 4a) and between E and θ_0 controlling for load percentage (Model 4b). We predicted inverse relationships between lordosis and both variables k and E according to Equation 16.

Statistical analyses

Data processing and analyses were performed using R statistical software (version 3.1.1). Variables were examined for normality using a Shapiro–Wilk test. GLMMs were analyzed using the “nlme” package in R (Pinheiro et al., 2012), and model coefficients were estimated using maximum likelihood. To account for repeated measures, each participant was identified by a unique number, which was used as the random grouping effect in GLMMs. When load percentage was included as a model covariate, each load condition was treated as a different factor level of the same variable to test for differences between trials. The proportion of variance (R^2) explained by GLMMs was estimated following Nakagawa and Schielzeth (2013) and Johnson (2014). To account for the hierarchical structure of the variance associated with fixed versus random effects, the “piecewiseSEM” package was used to describe marginal R^2 as the proportion of variance explained by fixed effects, and conditional R^2

described the proportion of variance explained by both fixed and random effects. Lordosis showed a hyperbolic relationship with both stiffness and elastic modulus (Equation 16), so log-log plots were used to analyze the relationship between lordosis and both bending stiffness and modulus. No alpha level was set for statistical significance, and instead effect sizes are discussed below with associated p-values.

Results

Table 3.3: Summary of the means (sd) of applied load and lumbar deformation variables across loading trials.

Loading trial	Load, kg	θ , deg	$\Delta\theta$, deg	ϵ	$\Delta\epsilon$
% Body Mass	Mean (sd)	Mean (sd)	Mean (sd)	Mean (sd)	Mean (sd)
0%	-	45.4 (16.1)	-	-0.0600 (0.0145)	-
5%	3.3 (0.6)	46.3 (15.7)	0.9 (3.8)	-0.0610 (0.0144)	-0.0010 (0.0043)
10%	6.6 (1.1)	45.8 (16.3)	0.4 (4.5)	-0.0601 (0.0148)	-0.0002 (0.0053)
15%	9.8 (1.7)	45.7 (16.3)	0.2 (4.3)	-0.0601 (0.0155)	-0.0002 (0.0051)
20%	13.1 (2.3)	44.4 (17.0)	-0.6 (7.2)	-0.0584 (0.0163)	0.0011 (0.0081)
25%	16.4 (2.8)	43.8 (17.0)	-1.2 (6.8)	-0.0575 (0.0162)	0.0017 (0.0078)
30%	19.7 (3.4)	43.5 (15.8)	-1.4 (8.2)	-0.0576 (0.0156)	0.0016 (0.0088)

The relationship between lordosis and applied load was highly variable between study participants (Figures 3.2), but the overall pattern of deformation indicated that lordosis tended to straighten under load. Mean θ decreased an average of 4.2% from 45.4° during the 0% trial to 43.5° during the 30% trial, and $\Delta\theta$ showed an average 1.4° decrease in mean curvature for that same interval (Table 3.3). Accounting for differences in the initial curved structure of the beam, measures of strain mirrored patterns of lordosis across loading trials, but with lower values for ϵ corresponding to greater angles of θ (Figure 3.3). Mean ϵ during the unloaded trial was measured at -0.06 (Table 3.3), indicating that unloaded lordosis resulted in mean bending strains of 6% flexion measured at the inner surface of the beam's curvature. Across loading trials, average ϵ increased in forward flexion of the spine by 3.3% resulting in a mean strain of -0.058 during the 30% loading condition (Table 3.3).

Testing **H1**, Model 1a revealed an association between mean θ and applied load mass such that each kilogram of added mass was associated with a 0.08° decrease in θ ($p = 0.05$, Table 3.4). Comparing relative changes in lordosis and load, Model 1b showed no differences in mean $\Delta\theta$ from 0% through 20% added mass, but $\Delta\theta$ decreased an average of 1.0° in the 25% load condition, though this was not significant at conventional p -values ($p = 0.06$). The 30% condition showed a 1.2° decrease ($p = 0.03$). Both models showed high levels of within-trial variability as the proportion of variance explained by fixed effects was less than 2% of the total variance in lumbar deformation, but when both fixed and random effects were considered, conditional R^2 values were 0.95 and 0.57 for Models 1a and 1b, respectively, suggesting the majority of lumbar deformation was explained by participant-specific random variations (Table 3.4).

Table 3.4: Summary of GLMMs testing **H1**, which predicts that lumbar spines tend to straighten under load.

Model 1a: Mean θ versus applied load				Model 1b: $\Delta\theta$ versus load condition			
Variable	Coef.	S.E.	P-value	Variable	Coef.	S.E.	P-value
Intercept	46.02	3.16	<0.001	Intercept (5%)	0.88	1.20	0.46
Load mass (kg)	-0.08	0.04	0.05	10%	-0.52	0.96	0.59
				15%	-0.63	0.96	0.51
				20%	-1.49	0.97	0.13
				25%	-1.87	0.97	0.06
				30%	-2.12	0.97	0.03
Marginal $R^2 = 0.001$				Marginal $R^2 = 0.02$			
Conditional $R^2 = 0.95$				Conditional $R^2 = 0.57$			

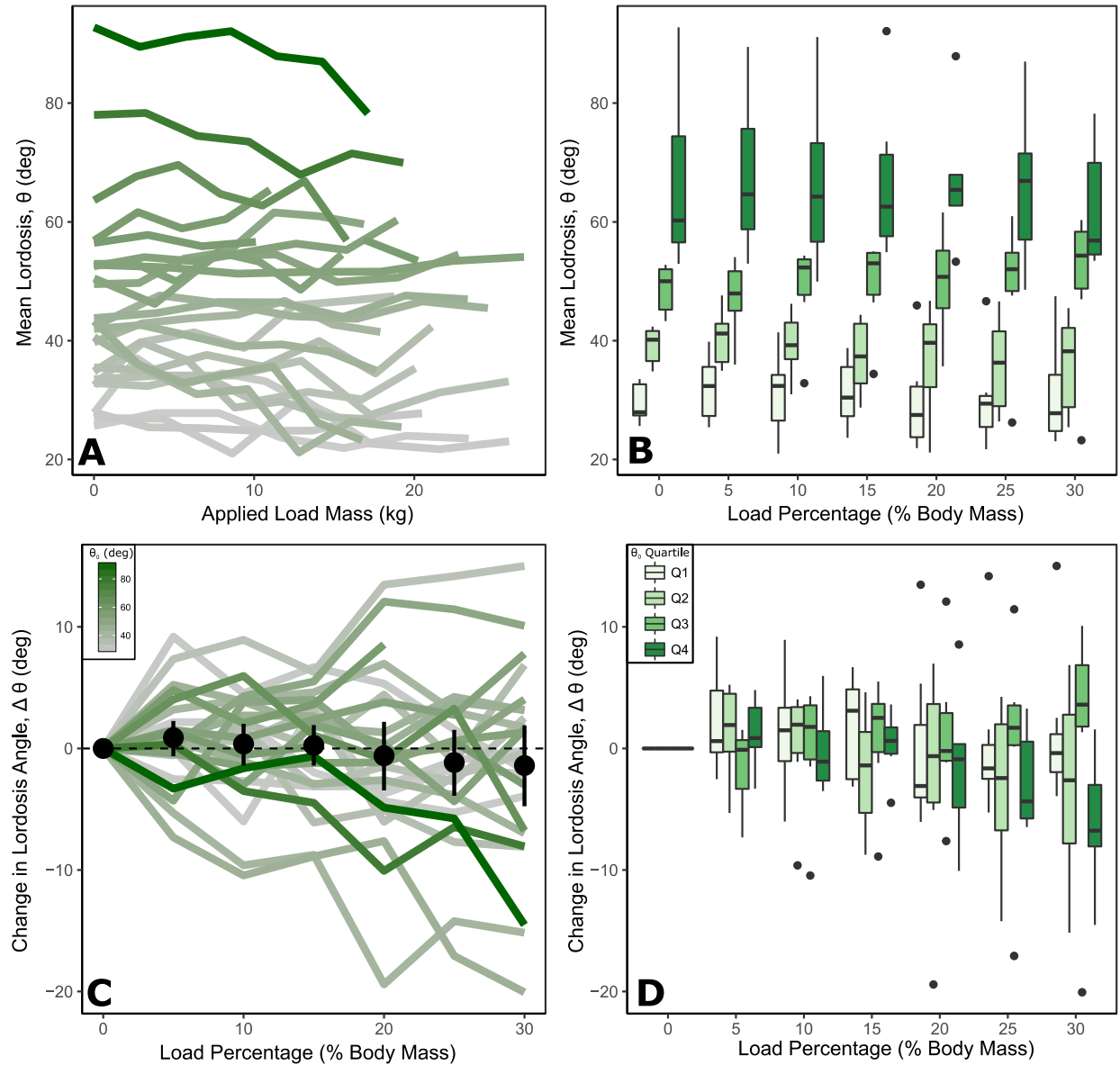


Figure 3.2: (A) Individual changes in mean lordosis with increased load mass across static loading trials. (B) Boxplots of mean lordosis versus load condition as a percentage of body mass showing variations in patterns of loading according to unloaded lordosis quartiles. (C) Individual relative changes in mean lordosis across loading trials as a percentage of body mass. Overall mean and 95% CI are shown as the black dots with error bars. (D) Boxplots of mean relative lordotic change versus load condition showing variations in patterns of loading according to unloaded lordosis quartiles. For all panels, darker green colors represent higher unloaded lordosis angles.

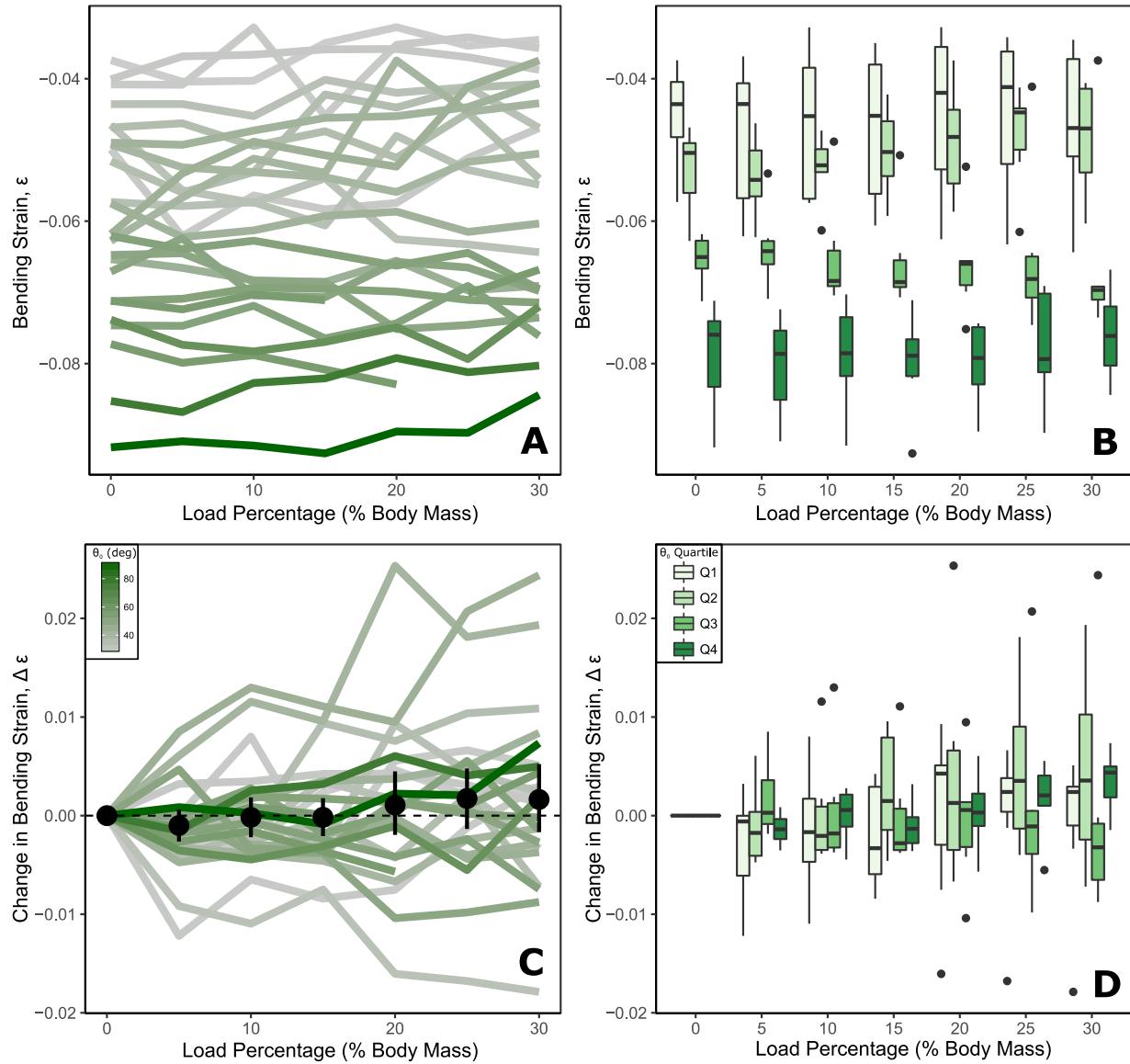


Figure 3.3: (A) Individual changes in bending strain with increased load mass across static loading trials. Note that negative strain values correspond with increased curvature in the loaded beam relative to curvature at the neutral axis. (B) Boxplots of mean strain versus load condition as a percentage of body mass showing variations in patterns of loading according to unloaded lordosis quartiles. (C) Individual relative changes in mean strain across loading trials as a percentage of body mass. Overall mean and 95% CI are shown as the black dots with error bars. (D) Boxplots of mean lordotic change versus load condition showing variations in patterns of loading according to unloaded lordosis quartiles. For all panels, darker green colors represent higher unloaded lordosis angles.

Table 3.5: Summary of GLMMs testing **H3**, which predicts that mean θ is associated with time-dependent changes in lordosis.

Model 2a: Within-trial variation in θ versus θ_0				Model 2b: Within-trial $\Delta\theta/\Delta t$ versus θ_0			
Variable	Coef.	S.E.	P-value	Variable	Coef.	S.E.	P-value
Intercept	0.58	0.34	0.09	Intercept	-1.58	0.67	0.02
5%	-0.12	0.23	0.61	5%	0.69	0.51	0.17
10%	-0.18	0.23	0.43	10%	-0.02	0.51	0.96
15%	-0.03	0.23	0.89	15%	-0.11	0.51	0.82
20%	0.38	0.23	0.10	20%	0.24	0.51	0.64
25%	0.39	0.23	0.10	25%	-0.08	0.51	0.88
30%	0.28	0.23	0.23	30%	0.63	0.51	0.22
Mean θ (deg)	0.02	0.01	0.01	Mean θ (deg)	0.03	0.01	0.01
Marginal $R^2 = 0.12$				Marginal $R^2 = 0.09$			
Conditional $R^2 = 0.32$				Conditional $R^2 = 0.21$			

Testing the second hypothesis (**H2**), that time-dependent patterns of lumbar deformation are predicted by lumbar posture, more curved lumbar lordosis was found to be associated with greater spinal instability over time. Within-trial variation in θ was positively associated with mean θ during a trial (Figure 3.4A; Table 3.5). Model 2a revealed that a 1.0° increase in mean θ was associated with a 0.02° increase in within-trial standard deviation of θ ($p = 0.01$, Table 3.5). Furthermore, curved lumbar spines tended to increase in curvature and straighter spines tended to decrease in curvature over time (Figure 3.4B). Model 2b showed that 1.0° increase in mean θ was associated with a $0.03^\circ/\text{min}$ increase in the slope of $\Delta\theta/\Delta t$ ($p = 0.01$, Table 2.6). This pattern of change in lordosis over time is further illustrated in Figure 3.5. Time-dependent changes in lordosis also showed no differences between loading conditions ($p > 0.10$, Table 3.5). Similar to Model 1, Models 2a and 2b showed that only a small percentage of total variance (9-12%) was explained by fixed effects compared to a larger percentage of variance explained by both random and fixed effects (21-32%) (Table 3.5).

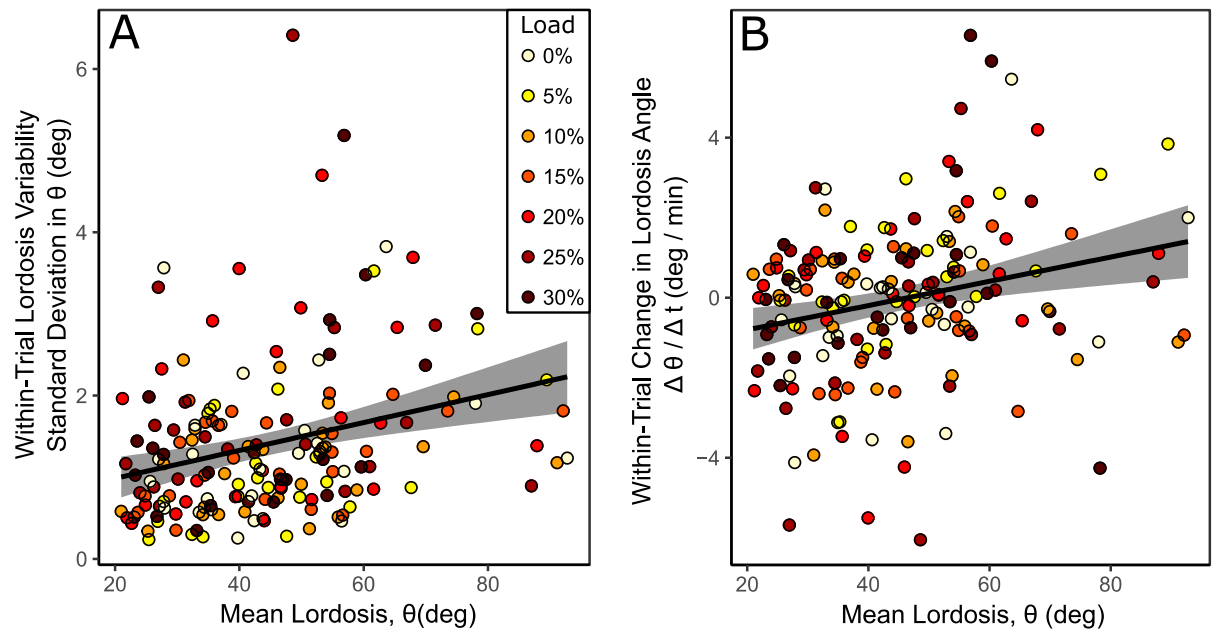


Figure 3.4: (A) Within-trial standard deviations in lordosis versus mean lordosis during the trial. The regression for Model 2a is shown as the solid black line with 95% confidence limits shaded. (B) The slope of the change in lordosis over time measured within each trial compared to the mean lordosis during that trial. The regression for Model 2b is shown as the solid black line with 95% confidence limits. For panels A and B, differences in color indicate the percent mass used during that loading trial. In both models, load condition was not a strong predictor time-dependent changes in lordosis.

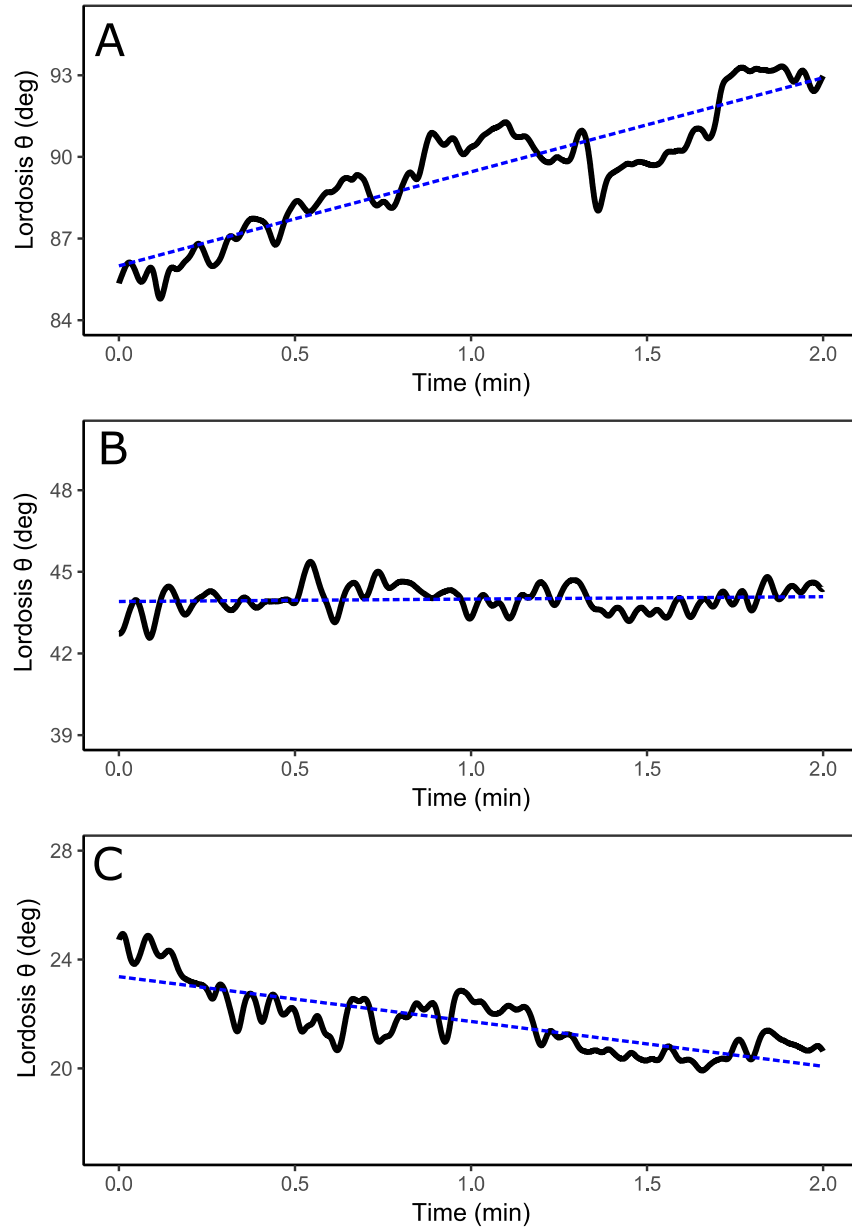


Figure 3.5: The pattern of change in lordosis over a 2-minute loading trial for three participants. (A) Participant 24, who had a high mean lordosis of 89.5° during the 5% loading trial, showed a positive change in lordosis of $3.8^{\circ} / \text{min}$. (B) Participant 10, who had an average lordosis of 44.0° during the 20% loading trial, showed almost no change in lordosis over time ($0.01^{\circ} / \text{min}$). (C) Participant 6, who had a low mean lordosis of 21.7° during the 25% loading trial, showed a decrease in lordosis of $1.8^{\circ} / \text{min}$.

The relationship between bending moments and lordosis change across trials showed a unique pattern of response for each individual (Figure 3.6). Variations in body mass, applied load mass, and trunk geometry led to variations in initial bending moments between participants (Figure 3.6A-B). Moments ranged between 41.8 and 200.5 N · m with a mean of 91.6 ± 36.5 N · m. The pattern of change in $\Delta\theta$ relative to ΔM varied across trials, as some individuals tended to show uniform increases or decreases in $\Delta\theta$ whereas others showed no pattern in $\Delta\theta$, which varied around the unloaded zero value (Figure 3.6A). However, changes in the beam's radius of curvature led to variations in the length of the bending moment arm (Equations 8 and 9), so the overall pattern of load-displacement showed that increases in M were associated with decreases in $\Delta\theta$ (Figure 3.6A). Similarly, the association between stress and strain varied across individuals, especially in initial levels of bending stress (Figure 2.6C-D). Bending stress, σ , ranged between 4.8 and 16.8 MPa with an average of 8.6 ± 2.8 MPa. Although there were differences in the magnitudes of change in σ and ϵ between individuals (Figure 2.6C), normalized changes in σ and ϵ revealed a similar pattern where relative increases in σ were associated with relative increases in ϵ (Figure 2.6D).

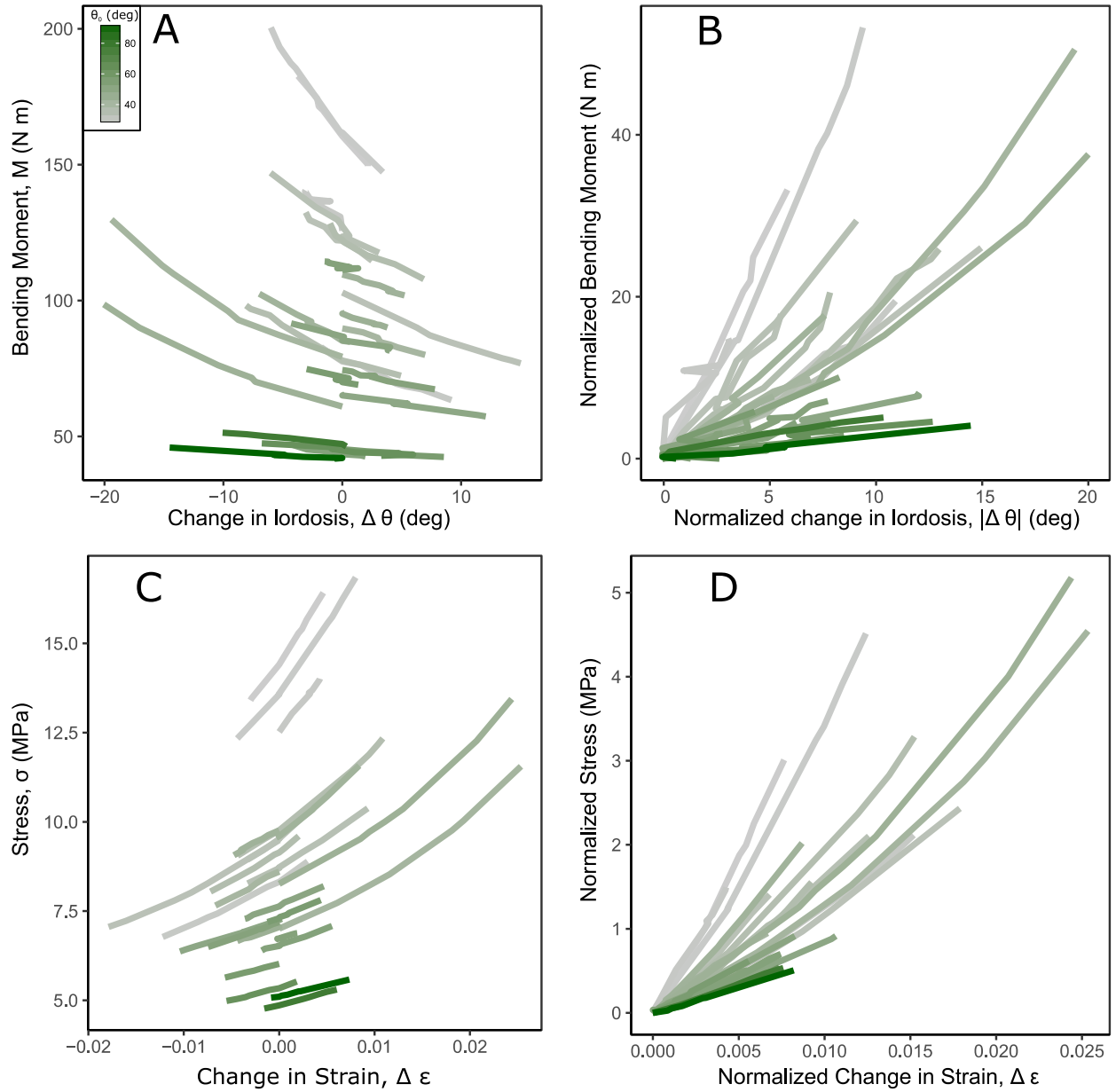


Figure 3.6: (A) Individual and (B) normalized differences in the relationship between lordosis versus total bending moments. (C) Individual and (D) normalized differences in bending strain versus total stress. Unloaded lordosis is indicated by darker green colors. Since participants varied in body mass and applied load mass, differences in moments between individuals were normalized in order to visualize differences in load-displacement and stress-strain curves. Moments were normalized to the minimum moment measured for each individual at zero. Changes in lordosis were standardized as absolute changes with minimum values at zero. The minimum values for each individual's stress and strain curve were set to zero. These plots illustrate differences in the patterns of lumbar deformation and strain as well as variations in the ranges of moments and stress. Although highly variable relative to unloaded positions, a clear pattern is seen such that individuals with more curved unloaded lumbar curvature (darker green) show shallower slopes than individuals with straighter lumbar spines (light grey).

Table 3.6: Summary of GLMMs testing **H3**, which predicts that stiffness and elastic modulus are inversely proportional to θ_0 .

Model 3a: Bending stiffness versus θ_0 and load				Model 3b: Elastic modulus versus θ_0 and load			
Variable	Coef.	S.E.	P-value	Variable	Coef.	S.E.	P-value
Intercept (5%)	3.67	0.45	<0.001	Intercept (5%)	4.60	0.31	<0.001
10%	0.05	0.08	0.50	10%	0.01	0.03	0.84
15%	0.03	0.08	0.70	15%	-0.03	0.03	0.36
20%	0.14	0.08	0.08	20%	0.04	0.03	0.29
25%	0.05	0.08	0.54	25%	0.02	0.03	0.61
30%	0.11	0.08	0.14	30%	0.04	0.03	0.18
$\log_{10} \theta_0$ (deg)	-2.20	0.27	<0.0001	$\log_{10} \theta_0$ (deg)	-1.52	0.19	<0.0001
Marginal $R^2 = 0.52$				Marginal $R^2 = 0.64$			
Conditional $R^2 = 0.64$				Conditional $R^2 = 0.83$			

Lumbar bending flexion stiffness, k , had a mean of $2.0 \pm 1.8 \text{ N} \cdot \text{m} / \text{deg}$, and elastic modulus, E , had a mean of $170.0 \pm 130.9 \text{ MPa}$. There were no strong differences in stiffness or modulus between trials ($p > 0.08$), suggesting that load-displacement and stress-strain curves were approximately linear (Table 3.6). When included as a covariate, sex also had no effect on the relationship between lordosis and bending modulus ($p = 0.38$) (Appendix Table 1.1; Appendix Figure 1.2). There was a strong negative correlation between k and θ_0 (Figure 3.7A-B). Model 3a revealed that a 1% difference in θ_0 was associated with a 2.2% decrease in k ($p < 0.0001$, Table 3.6). Within the range of θ_0 sampled among individuals in this study, this translates to a 10-fold difference in stiffness from the naturally straightest lordosis to the most curved lordosis. In Model 4b, which tested the association between E and θ_0 , there was a similar negative relationship between modulus and lordosis (Figure 7C-D). A 1% difference in θ_0 was associated with a 1.5% decrease in elastic modulus ($p < 0.0001$, Table 3.6), which translates to a 6.75-fold decrease in E from the naturally straightest to the most curved lordosis measured in this study. In both Models 3a and 3b, the marginal R^2 (fixed effects of θ_0 and load condition) explained 52% of the variance in k and 64% of the variance in E (Table 3.6), a higher proportion of variance than explained by fixed effects in Models 1 and 2.

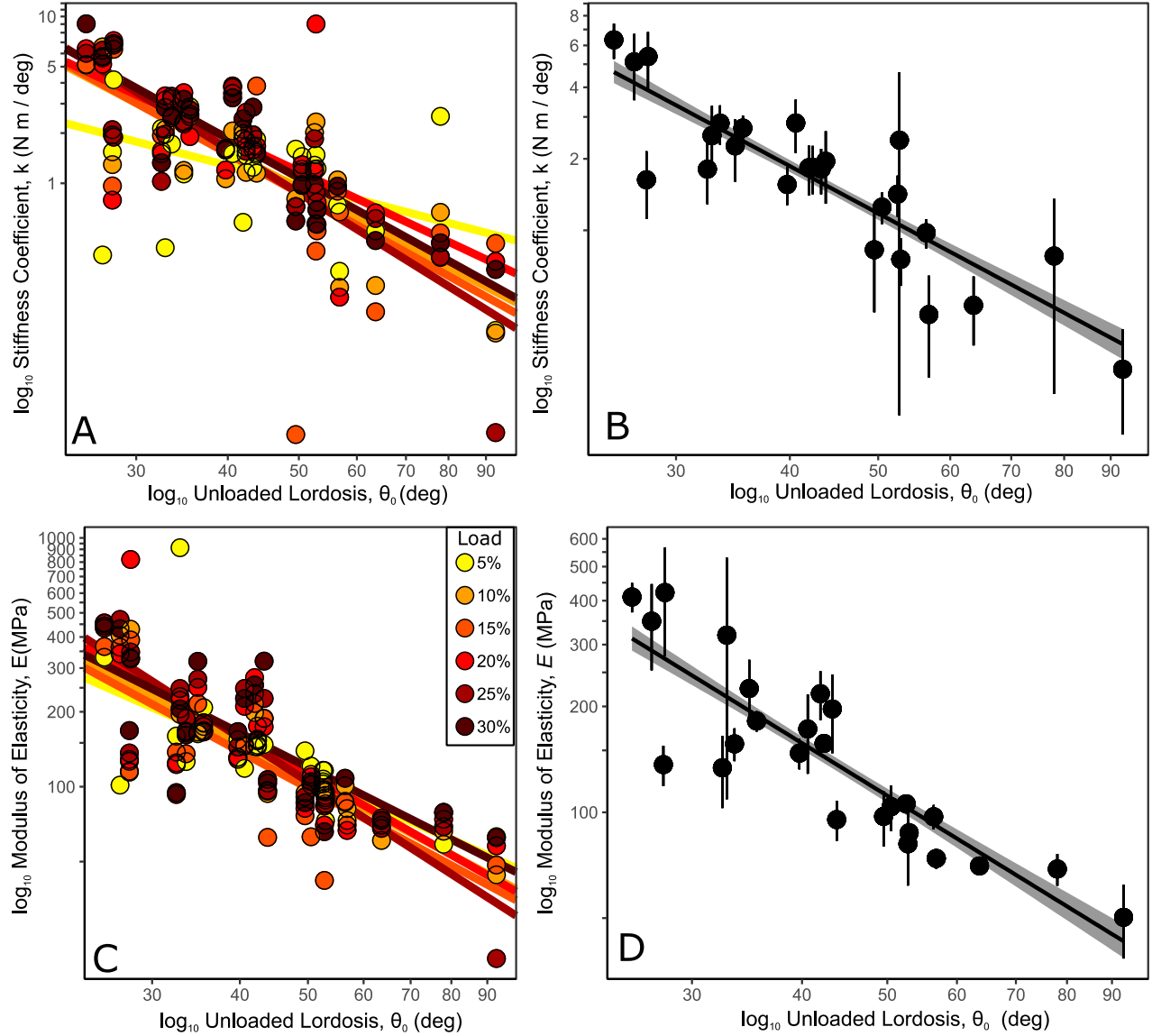


Figure 3.7: (A) Log-log relationship between mean unloaded lordosis versus bending stiffness measured across loading conditions, as predicted by Equation 16. (B) Relationship between mean unloaded lordosis versus mean bending stiffness for across individuals. (C) Relationship between mean unloaded lordosis versus elastic modulus measured across loading conditions. (D) Relationship between mean unloaded lordosis versus mean elastic modulus across individuals. For panels A and C, different loading conditions are represented by different colored points, and colored lines represent the slope of the relationship within loading conditions. Models 4a and 4b show that there were no differences in the slopes between loading conditions. For panels C and D, the overall regressions between lordosis and bending stiffness or elastic modulus from Models 3a and 3b (respectively) are shown as solid black lines with 95% confidence limits shaded.

Discussion

This study investigated how variations in lumbar lordosis affect patterns of spinal deformation during *in vivo* axial loading. Weight vest loading experiments tested increasing loads from 0-30% added mass in 5% increments. Internal differences in spinal structure from MRI scans were used to correct external measurements of spinal shape, and a beam bending model predicted bending stress and strain of the entire lumbar spine as a functional unit. Three hypotheses were tested to study how patterns of change in lordosis and bending strain relate to spinal stability and resistance to deformation within and between loading trials.

We found weak support for the first hypothesis (**H1**). Though lumbar spines showed an average tendency to straighten under load, the effect of load on lumbar posture was small. However, results are generally consistent with modeling studies that suggest straighter postures offer greater stability during loading (Scholten et al., 1988; Aspden, 1989; Shirazi-Adl and Parnianpour, 1999; Shirazi-Adl et al., 2002) as well as results reported from previous *in vivo* loading experiments (El-Rich et al., 2004; Meakin et al., 2008; Rodriguez-Soto et al., 2013). Our findings differ from previous work, however, in that we found higher degrees of variability in deformation and smaller differences in lordosis across loading trials. That said, direct comparison between studies is imprecise due to differences between methodologies. For example, Meakin et al. (2008) used positional MRI and multivariate shape analyses to study lumbar deformation during *in vivo* axial loading at 0, 8, and 16 kg loads (approximately 20% mean participant body mass). Their study found that each 8 kg increase in load was associated with 0.1 standard deviations of change toward straighter spinal postures. To test comparable measurements in the present study, we substituted Z-transformed variables into Model 1 and found that an 8kg applied load would lead to 0.11 standard deviations of predicted decrease in $\Delta\theta$, which is consistent with Meakin et al. (2008). In another study by Rodriguez-Soto et al. (2013), positional MRI was used to study Cobb angle

after applying a 50.8 kg load (70% mean body mass) to soldiers via a backpack for 45 min. These authors found an approximate 10° reduction in Cobb angle between loaded and unloaded conditions, nearly double the decrease in θ predicted in this study. However, Rodriguez-Soto et al. (2013) used a different measure of lumbar curvature (Cobb angle) that is correlated with the Central Angle method but does not capture as broad a range of variability in overall lumbar curvature (Castillo et al., 2017). Furthermore, loads used by Rodriguez-Soto et al. (2013) were nearly twice as heavy as the most massive loads used in this study (27 kg), and were applied for about 3 times longer than the total length of time of this experiment, likely explaining differences between results.

The results of this study supported **H2**, that differences in lumbar posture affect time-dependent changes in lordosis during loading. Over the course of a 2-min loading trial, more curved spines showed a relative increase in lordosis while straighter spines showed a relative decrease in lordosis, supporting findings reported by Meakin et al. (2008). We also found that straighter spines showed less variation in lordosis over time. Because dynamic *in vivo* changes in lordosis during loading have not been previously reported, this study provides novel evidence that lumbar postures affect time-dependent parameters related to spinal stability. Although these results may be explained by passive tissue creep between motion segments (e.g. Adams and Dolan, 1996), an alternative explanation may be that time-dependent changes in lordosis are the result of differences in the active recruitment of muscles. Further studies are needed to relate patterns of change in lordosis over time to the contributions of passive versus active structures by measuring muscle activation in the trunk stabilizers during axial spinal loading.

The most strongly supported hypothesis (**H3**) was that bending stiffness and elastic modulus are inversely related to natural variations in lordosis. We found that the lumbar spine's resistance to deformation showed a strong negative correlation with lordosis explaining 52% of the variation in

lumbar bending stiffness and 64% of the variation in elastic modulus. Although the relationship between lumbar curvature and elastic modulus was predicted by the model (Equations 14-16), support for **H3** provides important empirical evidence that lordosis affects the spine's resistance to static loading.

To evaluate the reliability of the beam bending model, lumbar stiffness and elastic modulus estimates were compared to values reported in the literature. Average lumbar bending stiffness in this study was estimated to be $2.0 \text{ N} \cdot \text{m/deg}$, which is consistent with mean stiffness coefficients reported from *ex vivo* studies of intervertebral segments in flexion ($2.0 - 2.6 \text{ N} \cdot \text{m/deg}$) tested throughout the thoracolumbar spine (Markolf, 1972; Panjabi et al., 1976; White and Panjabi, 1978). However, our estimates for bending stress appear to be much higher than other studies. We modeled stress and strain at the inner surface of the beam (i.e. the posterior border of the lumbar vertebral bodies) because *ex vivo* experiments suggest that straighter lumbar postures affect patterns of stress at the posterior annulus fibrosus (Adams and Hutton, 1985). Yet our estimate of mean stress (8.6 MPa) at the beam's inner surface is higher than the mean ultimate failure stress measured at the outer region of the annulus in isolated intervertebral discs ($7.39 \pm 4.25 \text{ MPa}$) (Skrzypiec et al., 2007). Though stress estimates reported here may appear unreasonably high, note that these estimates do not imply that the intervertebral discs were experiencing forces above failure; rather our model describes the total bending resistance of the lumbar spine attributed to multiple tissues including ligaments, tendons, bones, muscles, and discs. This is evident from our mean elastic modulus estimate of 170 MPa, which lies within the ranges of tensile elastic moduli reported for superficial (150-500 MPa) and deep (0-200 MPa) cartilages throughout the body (LeVeau, 1992). The maximum modulus measured in this study (913 MPa) is also within the lowest range of moduli reported for various tendons (800-2000 MPa), but far below tensile moduli of trabecular and cortical bone (6 - 18.4 GPa). This kind of variation in elastic modulus is expected from a model of the global bending behavior of the lumbar spine *in vivo*, where loading can be variably resisted by passive and active structures throughout lumbopelvic region.

Although this study provides crucial information regarding the relationship between lordosis and lumbar stability, there are several limitations. Most notably, we relied on a beam model to predict and estimate bending parameters. Beam models make several assumptions regarding the shape, density, and behavior of the lumbar spine. Such assumptions apply to other studies as well. Euler buckling models are frequently used to predict the shape of the human vertebral column (e.g. Meakin et al., 1996; Whitcome, 2012), but buckling models—which also assume a circular, solid structure to the spine’s cross section—are limited in their ability to account for bending moments and predict changes in lumbar curvature with respect to loading. Finite element models also make assumptions when determining the behavior of elements and parameters included in the model. Beam models, though highly simplified, offer a useful alternative to predict overall bending behavior of the loaded spine. These models have proven reliable for predicting spinal deformation in experimental studies of vertebrates (Gal, 1993; Long et al., 1997; Etnier, 2001) as well as in humans (Harrison et al., 2001; Castillo et al., 2017). Compared to buckling models, curved beam models also have the benefit of being able to account for structural differences in the position of the neutral bending axis. The geometric approximations used to estimate NA in this study almost certainly oversimplify a complex system, but have the advantage of providing *in vivo* estimates of lumbar stress and strain in relation to spinal shape. An additional limitation of this study is the reliance on external markers to measure spinal curvature. Despite some imprecision in this measurement, we attempted to incorporate MRI imaging to correct external measurements for internal variations in spinal shape. However, radiological imaging of the spine under load would greatly improve future studies. A final limitation is that our study population sampled only a limited range of physical activity levels, ages, or other factors that may have important influences on the resistance of the spine to lumbar spinal deformations. Studies of other populations—particularly those that include individuals who frequently carry heavy loads—would provide an interesting comparative reference for the bending parameters measured in this study.

In summary, this study offers insights into how the lumbar spine maintains structural stability. As both modeling and experimental studies have suggested, straighter lumbar spines offer several advantages compared to curved postures, including greater resistance to bending, increased stability, and reduced risk of injury since lordosis is associated with higher intervertebral shearing forces (Shirazi-Adl et al., 2002; Arjmand and Shirazi-Adl, 2005; Meakin et al., 2008). But these results raise a conundrum: if straighter spines offer more stability, greater resistance to bending, and lower levels of injury risk during loading, then why do humans show such variability in lordosis?

One potential explanation is that differences in unloaded lordosis covary with other parameters related to spinal stiffness such as muscle strength and passive tissue compliance, leading to tradeoffs. For example, although it may be advantageous for humans to have a straighter, stiffer, and less injury-prone spine, lumbar stability may come at the cost of decreased lower back mobility. In a previous study of the biomechanical factors related to natural variations in lordosis, we found an interaction between trunk muscle strength and spinal flexibility, which explains 65% of the variation in standing lordosis (Castillo et al., 2017). Naturally straighter spines were associated with less sagittal range of motion and relatively stronger abdominal muscles (compared to back muscles), while naturally curved spines were associated with greater ranges of motion and relatively stronger back muscles. However, controlling for both strength and flexibility, relative back muscle size also had a strong effect to limit lumbar range of motion. Together, covariation between spinal shape and factors such as trunk strength and lumbar range of motion have complex, competing effects on lumbar stability. While relatively stronger back muscles provide increased stiffness and stability, relatively stronger back muscles also increase the amount of lordosis, thus negating the advantages of having a straighter lumbar shape. Another tradeoff may arise due to the vital role of lumbar flexibility during locomotion. Changes in lordosis during locomotion are known to transfer energy between the upper and lower body (Gracovetsky and Iacono, 1987; Alexander, 1997), including shock forces. While straighter spines provide greater stiffness and

stability to support loads statically, reduced lordosis may also reduce the dynamic capacity of the lumbar spine to attenuate shock accelerations during running, when impact shocks are highest. This tradeoff between static structural stiffness and dynamic shock attenuation due to variations in lordosis may lie at the root of the competing evolutionary pressures acting on the hominin spine, a question explored in later chapters.

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References

- Abitbol MM. 1988. Effect of posture and locomotion on energy expenditure. *Am J Phys Anthropol* 77: 191–9.
- Adams MA, Dolan P. 1996. Time-dependent changes in the lumbar spine's resistance to bending. *Clin Biomech* 11(4): 194-200.
- Adams MA, Hutton WC. 1985. The effect of posture on the lumbar spine. *J Bone Joint Surg* 67(4): 625-9.
- Adams MA, Bogduk N, Burton AK, Dolan P. 2006. *The biomechanics of back pain*. Churchill Livingstone, Edinburgh.
- Alexander RM. 1997. Elasticity in human and animal backs. In: *Movement, Stability and Low Back Pain* Vleeming A, Mooney V, Dorman T. Snijders CJ, Stoeckart R, eds. Edinburgh, UK: Churchill Livingstone.
- Arjmand N, Shirazi-Adl A. 2005. Biomechanics of changes in lumbar posture in static lifting. *Spine* 30(23): 2637-48.
- Aspden RM. 1989. The spine as an arch: a new mathematical model. *Spine* 14(3): 266-74.
- Been E, Gómez-Olivencia A, Kramer PA. 2012. Lumbar lordosis of extinct hominins. *Am J Phys Anthropol* 147: 64–77.
- Botsford DJ, Esses SI, Ogilvie-Harris DJ. 1994. In vivo diurnal variation in intervertebral disc volume and morphology. *Spine* 19: 935-40.
- Bucciarelli LL. 2009. *Engineering mechanics for structures*. New York: Dover Publications.
- Castillo ER, Hsu C, Mair RW, Lieberman DE. 2017. Testing biomechanical models of human lumbar lordosis variability. *Am J Phys Anthropol* 00: 1–12. <https://doi.org/10.1002/ajpa.23189>
- Crisco JJ. 1989. *The biomechanical stability of the human lumbar spine: Experimental and theoretical investigations (Doctoral Dissertation)*. New Haven, CT: Yale University.
- Dempster WT. 1955. Space requirements of the seated operator: geometrical, kinematic, and mechanical aspects of the body with special reference to the limbs. WADC Technical Report (Wright-Patterson Air Force Base). 55-159.
- Dowling, NE. 2012. *Mechanical Behavior of Materials: Engineering Methods for Deformation, Fracture, and Fatigue (2nd Ed)*. New Jersey: Prentice Hall.
- El-Rich M, Shirazi-Adl A, Arjmand N. 2004. Muscle activity, internal loads, and stability of the human spine in standing postures: combined model and in vivo studies. *Spine* 29(23): 2633-42.
- Etnier SA. 2001. Flexural and torsional stiffness in multi-jointed biological beams. *Biol Bull* 200: 1-8.
- Gál J. 1993. Mammalian spinal biomechanics. I. Static and dynamic mechanical properties of intact intervertebral joints. *J Exp Biol* 174: 247–80.
- Gardner-Morse MG, Stokes IA. 2004. Structural behavior of human lumbar spinal motion segments. *J Biomech* 37(2):205-12.
- Gracovetsky SA, Iacono S. 1987. Energy transfers in the spinal engine. *J Biomed Eng* 9: 99–114.
- Hanavan EP. 1964. A mathematical model of the human body. AMRL-TR-64-102. AMRL TR. 1–149.

- Harrison DE, Harrison DD, Janik TJ, Jones EW, Cailliet R, Normand M. 2001. Comparison of axial and flexural stresses in lordosis and three buckled configurations of the cervical spine. *Clin Biomech* 16(4): 276-84.
- Janevic J, Ashton-Miller JA, Schultz AB. 1991. Large compressive preloads decrease lumbar motion segment flexibility. *J Orthop Res* 9(2): 228-36.
- Johnson PC. 2014. Extension of Nakagawa & Schielzeth's R2GLMM to random slopes models. *Methods Ecol Evol* 5(9): 944-6.
- Lampl M. 1992. Further observations on diurnal variation in standing height. *Ann Hum Biol* 19: 87–90.
- LeVeau BF. 1992. *Biomechanics of human motion: Basics and beyond for the health professions* (3rd ed.) Philadelphia: W.B. Saunders.
- Lin HS, Liu YK, Adams KH. 1978. Mechanical response of the lumbar intervertebral joint under physiological (complex) loading. *J Bone Joint Surg Am* 60(1):41-55.
- Long JH, Pabst DA, Shepherd WR, McLellan, WA. 1997. Locomotor design of dolphin vertebral columns: bending mechanics and morphology of *Delphinus delphis*. *J Exp Biol* 200: 65–81.
- Lovejoy CO, McCollum MA. 2010. Spinopelvic pathways to bipedality: why no hominids ever relied on a bent-hip-bent-knee gait. *Philos Trans R Soc Lond B Biol Sci* 365: 3289–99.
- Markolf KL. 1972. Deformation of the thoracolumbar intervertebral joints in response to external loads. *J Bone Joint Surg* 54(3): 511-33.
- Meakin JR, Hukins DW, Aspden RM. 1996. Euler buckling as a model for the curvature and flexion of the human lumbar spine. *Proc Biol Sci* 263: 1383–7.
- Meakin JR, Smith FW, Gilbert FJ, Aspden RM. 2008. The effect of axial load on the sagittal plane curvature of the upright human spine in vivo. *J Biomech* 41: 2850–4.
- Nakagawa S, Schielzeth H. 2013. A general and simple method for obtaining R2 from generalized linear mixed-effects models. *Methods in Ecol and Evol* 4(2): 133-42.
- Panjabi MM, Crisco JJ, Vasavada A, Oda T, Cholewicki J, Nibu K, Shin E. 2001. Mechanical properties of the human cervical spine as shown by three-dimensional load–displacement curves. *Spine* 26(24): 2692-700.
- Patwardhan AG, Havey RM, Meade KP, Lee B, Dunlap B. 1999. A Follower Load Increases the Load-Carrying Capacity of the Lumbar Spine in Compression. *Spine* 24(10): 1003-9.
- Pinheiro J, Bates D, DebRoy S, Sarkar D and R Core Team (2017). *nlme: Linear and Nonlinear Mixed Effects Models*. R package version 3.1.1.
- Rodriguez-Soto AE, Jaworski R, Jensen A, Niederberger B, Hargens AR, Frank LR, Kelly KR, Ward SR. 2013. Effect of load carriage on lumbar spine kinematics. *Spine* 38(13): E783-91.
- Saha D, Gard S, Fatone S, Ondra S. 2007. The effect of trunk-flexed postures on balance and metabolic energy expenditure during standing. *Spine* 32: 1605–11.
- Scholten PJ, Veldhuizen AG, Grootenboer HJ. 1988. Stability of the human spine: a biomechanical study. *Clin Biomech* 3(1): 27-33.

- Schwab F, Lafage V, Boyce R, Skalli W, Farcy JP. 2006. Gravity Line Analysis in Adult Volunteers. *Spine* 31: E959-67.
- Shirazi-Adl A, Parnianpour M. 1999. Effect of changes in lordosis on mechanics of the lumbar spine-lumbar curvature in lifting. *J Spinal Disord* 12(5): 436-47.
- Shirazi-Adl A, Sadouk S, Parnianpour M, Pop D, El-Rich M. 2002. Muscle force evaluation and the role of posture in human lumbar spine under compression. *Eur Spine J* 11(6): 519-26.
- Skrzypiec D, Tarala M, Pollintine P, Dolan P, Adams MA. 2007. When are intervertebral discs stronger than their adjacent vertebrae?. *Spine* 32(22): 2455-61.
- Strickland AL, Shearin RB. 1972. Diurnal height variation in children. *J Pediatr* 80: 1023-5.
- Syczewska M, Öberg T, Karlsson D. 1999. Segmental movements of the spine during treadmill walking with normal speed. *Clin Biomech* 14(6): 384-8.
- Tillmann V, Clayton PE. 2001. Diurnal variation in height and the reliability of height measurements using stretched and unstretched techniques in the evaluation of short-term growth. *Ann Hum Biol* 28: 195–206.
- Vette AH, Yoshida T, Thrasher TA, Masani K, Popovic MR. 2011. A complete, non-lumped, and verifiable set of upper body segment parameters for three-dimensional dynamic modeling. *Med Eng Phys* 33(1): 70-9.
- Voss LD, Bailey BJ. 1997. Diurnal variation in stature: is stretching the answer? *Arch Dis Child*. 77: 319–22.
- Whitcome KK, Shapiro LJ, Lieberman DE. 2007. Fetal load and the evolution of lumbar lordosis in bipedal hominins. *Nature* 450: 1075–8.
- Whitehouse RH, Tanner JM, Healy MJ. 1974. Diurnal variation in stature and sitting height in 12-14-year-old boys. *Ann Hum Biol* 1: 103–6.

Chapter 4 – Effects of lordosis on shock attenuation in the human lumbar spine during walking and running

Introduction

Lumbar lordosis is an essential feature of the human vertebral column that facilitates normal spinal function and helps maintain a balanced trunk orientation. However, fossil evidence indicates there has been considerable variation in lordosis within modern human populations and among fossil hominins (Been et al., 2012), suggesting that variations in lumbar curvature may serve other functions. Additionally, there are marked differences in lumbar posture among different kinds of athletes. For example, sprinters, long distance runners, and soccer players tend to have more curved lumbar spines, but body builders and swimmers—athletes who do not experience repetitive impacts and/or employ high levels of isometric contractions in the upper body —tend to have much straighter lumbar spines (Uetake and Ohtsuki, 1993; Wodecki et al., 2002). Thus, one hypothesized function of lordosis is that variations in spinal curvature modulate impact-related shocks transmitted through the vertebral column during locomotion. Although motions of the lumbar spine are known to transfer energy between the upper and lower body during gait (Gracovetsky and Iacono, 1987; Syczewska et al., 1999; Grasso et al., 2000), and intervertebral discs are considered important “shock absorbers” within the human body (Voloshin et al., 1981, 1998; Voloshin and Wosk, 1982; Adams and Hutton, 1985; Adams et al., 2006), little is known about how lordosis affects shock accelerations propagated through the spine during locomotion. Therefore, this study investigates the relationship between lordosis variability and impact-related shock transmission through the lumbar spine during walking and running.

Studies suggest a relationship between lordosis and kinematic differences in gait. Individuals with reduced lordosis tend to use slower walking speeds and shortened strides, and individuals with greater pelvic tilt and presumably higher lordosis (Vialle et al., 2005) tend to use longer strides when

running (Grasso et al., 2000; Sarwahi et al., 2002; Hirose et al., 2004; Franz et al., 2009). Longer strides are also associated with greater reliance on rearfoot strike patterns and reduced leg stiffness, both characteristics hypothesized to increase impact forces and injury risk (Lieberman et al., 2010; Perl et al., 2012; Davis et al., 2015). Associations between lordosis, stride length, limb compliance, and foot strike likely affect shock accelerations transmitted through the musculoskeletal system.

During locomotion the rapid deceleration of the body at foot contact results in an impact-related shock wave, which propagates up from the ground through the body until reaching the head (Voloshin et al., 1981, 1998; Voloshin and Wosk, 1982; Shorten and Winslow 1992; Hamill et al. 1995; Derrick et al. 1998; Mercer et al., 2002; Edwards et al., 2012; James et al., 2014; Gruber et al., 2014). Impact peaks can be substantial with magnitudes between 0.6 and 1.0 times body weight (BW) during walking and 1.0 to 3.0 times BW during running (Nigg et al., 1995; Whittle, 1999). Furthermore, impact loading rates can be as high as 500 BW / s when running barefoot using a rearfoot strike pattern (Lieberman et al., 2010). Attenuation of these large, rapid impact peaks—which are unique to humans and not consistently observed during chimpanzee locomotion (Pontzer et al. 2014)—is crucial because the resulting impulse can disrupt the vestibular-ocular reflex and potentially lead to injury and pathology (Pozzo et al. 1990, 1991; Whittle 1999).

It is important to consider not only shock magnitude but also the frequency range within which shock waves travel. Shock accelerations during locomotion are composed of three main frequency components, which occur simultaneously within the time domain but propagate differently through the body (Shorten and Winslow, 1992). Low-frequency components typically fall between 4 and 10 Hz and are the result of active motion rather than impact (Shorten and Winslow, 1992; Angeloni et al., 1994; Hamill et al., 1995). Low-frequency signals are therefore often filtered to avoid biased estimates of impact shock (James et al., 2014). The high-frequency component is also unrelated to impact, typically

occurs over 30 Hz, and represents the resonant frequency of the inertial device, which varies based on the sensor's mass, method of mounting, and underlying soft tissue movements (see Forner-Cordero et al., 2008). However, the mid-frequency component represents the critical frequency range related to impacts and injury (Shorten and Winslow 1992; Hamill et al. 1995; James et al., 2014). The mid-frequency component typically occurs between 10 and 20 Hz and is similar for barefoot and shod running (Bobbert et al., 1991; Shorten and Winslow, 1992; Derrick et al., 1998; Mercer et al., 2002; Hamill et al., 1995; Edwards et al., 2012; Gruber et al., 2014; Giandolini et al., 2016) but is slightly higher at approximately 22 Hz for barefoot walking compared to shod (18 Hz), though it is unclear why (James et al., 2014).

Impact-related shocks can be attenuated in two main ways: passive attenuation via soft tissues, footwear, and ground substrate compliance; or active attenuation via muscle activity or modifications to lower limb kinematics (Paul et al., 1978; McMahon et al., 1987; Derrick et al., 1998; Whittle, 1999; Edwards et al., 2012; Addison and Lieberman, 2015). But previous work has not directly addressed the question of whether spinal shape affects lumbar shock attenuation. Existing studies of spinal shock attenuation have mainly relied on measurements from accelerometers mounted far away from the spine (e.g. the lower limb and head) and analyses of only acceleration peaks in the time domain rather than the frequency domain. For example, Voloshin and Wosk (1982) found that walking individuals with lower back pain have reduced capacity for attenuation of the peak axial accelerations measured at the medial femoral condyle and head. In another study, Dimitriadis et al. (2011) found that individuals running for one hour exhibited a 4° average reduction in static sitting lordosis and decreased average lumbar disc height, which may suggest reduced shock attenuation related to disc height and lumbar posture. In addition, several studies have explored the effects of foot strike patterns and footwear on impact shocks through the musculoskeletal system (Ogon et al., 2001; Divert et al., 2005; Lieberman et al., 2010; Kulmala et al., 2013; Boyer et al., 2014; Giandolini et al., 2016). One such study found that

runners using a rearfoot rather than forefoot strike pattern showed an average reduction in dynamic lumbar range of motion of 1.2° and 30% greater attenuation of the peak axial accelerations measured at the tibia and head (Delgado et al, 2013).

If lordosis influences lumbar shock attenuation, then the mechanism driving this association is most likely due to the lumbar spine's viscoelastic properties. On the one hand, Rak (1993) speculated that lordosis allows the spine to act like a “shock absorber,” perhaps explaining differences in lordosis among fossil hominins. This hypothesis predicts that spinal curvature may serve to dissipate energy associated with shock impacts as bending and rotational deformations rather than pure axial compression. On the other hand, Adams et al. (2006) hypothesized that the principal function of human spinal curvature is to absorb and release energy elastically during locomotion. Dynamic motions of the lumbar spine in flexion and extension oscillate approximately 1-2° in amplitude for each lumbar vertebral level during walking (Syczewska et al., 1999), perhaps indicating spring-like behavior during locomotion to load and unload the spine. However, Adams et al. (2006) further speculated that spinal tissues themselves are unable to store large amounts of strain energy. The intervertebral discs appear to be inadequately vascularized to dissipate heat effectively, so viscoelastic function of the spine is most likely actively aided by trunk muscles and passively by tendons to cope with dynamic impacts (Alexander, 1997; Adams et al., 2006). Thus, it is unclear whether the lumbar spine functions to attenuate impact shocks, and if so which tissues contribute to attenuation.

Hypotheses

This study tests three hypotheses. The first hypothesis (**H1**) tests whether variations in lordosis influence the shock attenuation capacity of the lumbar spine. We predict that individuals with increased lordosis show greater capacity for lumbar shock attenuation during walking and running compared to individuals with straighter backs. The second hypothesis (**H2**) addresses the mechanism by which lumbar

shock attenuation may occur. We predict that differences in the dynamic motion of the lumbar spine during gait reflect variations in the viscoelastic properties of the lower back. Assuming the lumbar spine behaves like an Euler-Bernoulli beam containing elastic and viscous elements, we predict that the spine's response to shock vibrations will follow the Kelvin-Voigt generalization such that elasticity is proportional to displacements and damping is proportional to beam angular displacement velocity (Herrmann, 2008). If the lumbar spine exhibits these viscoelastic properties, then **H2** predicts that impact-related shock attenuation is positively associated with the amplitude of lordosis angular displacements but negatively correlated with the rate of lordosis angular displacements. To further explain the relationship between lordosis and lumbar shock attenuation, the third hypothesis (**H3**) tests whether variations in shock attenuation are more strongly predicted by intervertebral discs or by variations in lumbar posture. Intervertebral discs are often considered the primary passive “shock absorbers” of the spine (Voloshin et al., 1981, 1998; Voloshin and Wosk, 1982; Alexander, 1997; Adams et al., 2006). However, discs account for the majority of the variation in lumbar curvature among adults (Shefi et al., 2013), suggesting that intervertebral disc morphology is correlated with lumbar spinal shape. To test **H3**, we predict that lordosis remains a strong predictor of shock attenuation after accounting for covariation between intervertebral disc height and lumbar curvature.

Materials and Methods

Participants

Study participants were recruited from the greater Boston area for a series of experiments on the lumbar spine. These studies were conducted on different days over the course of four months. To reduce potential degenerative changes in spinal posture that can occur among older adults (Schwab et al., 2006), only young adults aged 18-35 years were recruited. Twenty-seven participants (14 male, 13 female) completed the study. Participants sampled a range of heights and body masses (Table 4.1). A

general health questionnaire was administered prior to experiments, and participants were excluded if they had a history of back pain, sciatica, scoliosis, major illness, or injury in the last three months that could compromise gait. Written informed consent was given for the study, which was approved by the Committee on the Use of Human Subjects at Harvard University. All experimental protocols took place between 3 and 6 PM to minimize circadian effects of viscoelastic creep in spinal tissues (Strickland and Shearin, 1972; Whitehouse et al., 1974; Lampl, 1992; Botsford et al., 1994; Voss and Bailey, 1997; Tillman and Clayton, 2001).

Imaging

Magnetic resonance imaging was conducted at the Center for Brain Science Neuroimaging Facility of Harvard University using a Siemens TIM Trio (3-T) scanner. Imaging protocols are described in Castillo et al. (2017). Briefly, a standard spine-array and large-flex coils were used to scan participants in a neutral, supine position with legs extended, arms resting at their sides. A midsagittal, single-slice “localizer” scan (TR = 8.6 ms, TE = 4 ms; 7 mm thickness, 1.7 mm/pixel) was used to orient individuals in the scanner, and from this scan the craniocaudal maximum disc height (MDH) was measured for each intervertebral level from T12-L1 through L5-S1 using ImageJ (National Institutes of Health). Average disc height was standardized for body size by calculating the relative maximum disc height (relMDH) as the cube of MDH divided by body mass to account for scaling.

Procedures

Study participants were barefoot during experiments in order to reduce shock attenuation from passive structures such as shoes (Paul et al., 1978; Whittle, 1999; Addison and Lieberman, 2015), and participants were instructed to use a rearfoot strike pattern during running trials in order to maximize impact-related shocks that reach the spine and to control for variation caused by different strike types (Ogon et al., 2001; Lieberman et al., 2010; Delgado et al., 2013). After being instrumented with

accelerometers and motion tracking markers (see *Kinematics* below), participants stood motionless in a neutral position (arms resting comfortably at their sides) for 2 min while 3-D kinematics measured static standing posture. After the standing trial, participants walked and ran on an instrumented treadmill with embedded force plates (1000 Hz; Bertec, OH, USA) at comfortable Froude numbers of 0.25 and 1.00. Speed was made dimensionless and standardized to lower limb length to account for differences in body size (Alexander and Jayes, 1983) such that:

Equation 1
$$Froude\ number = \frac{v^2}{gl}$$

where v is velocity, g is gravitational acceleration, and l is limb length from greater trochanter to the ground. Absolute speed averaged 1.53 ± 0.16 m/s for walking and 3.00 ± 0.18 m/s for running. Each trial lasted 2 minutes during which 30 seconds of accelerometry and kinematic data were simultaneously collected approximately midway through the trial.

Accelerometers

Two tri-axial piezoelectric accelerometers (Endevco model 35A, San Juan Capistrano, CA) were secured to rectangular pieces of aluminum (21.5 x 14.0 x 0.5 mm) using cyanoacrylate. The total mass of each sensor (including the aluminum mount) was 2.7 grams. Accelerometers were affixed firmly using adhesive tape to the skin overlying the T11-T12 and S1-S2 intervertebral levels. These sites were chosen to isolate shock attenuation within the lumbar vertebral column, measuring incoming shocks from the ground at the lumbosacral joint (i.e. “low-back”) and outgoing impact shocks at the thoracolumbar joint (i.e. “mid-back”). The vertical (Z) axes of accelerometers were aligned with the spine’s craniocaudal axis, and the transverse (Y) axes were aligned with the body’s dorsoventral axis. Sensors were powered and amplified by an Isotron signal conditioner (Endevco model 2793, San Juan Capistrano, CA) that passed signal sampled at 1000 Hz to a common analog-to-digital converter board, which synchronized with

kinematic data. To reduce soft tissue oscillations, skin laterally adjacent to each accelerometer was pre-loaded by manually stretching and taping the skin perpendicular to the spine's craniocaudal axis using kinesiology tape. We chose to pre-load the skin with tape and use low-mass accelerometers to increase the stiffness of the attachment between the sensor and the skin. These methods have been shown to be effective for reducing motion artifact due to oscillations of the inertial sensor (Saha and Lakes, 1977; Ziegert and Lewis, 1979; Nokes et al., 1984; Trujillo and Busby, 1990; Forner-Cordero et al., 2008).

To remove the potentially error-prone low-frequency accelerations and higher frequency noise (James et al., 2014), signal data were filtered using a second-order, zero-phase digital Butterworth filter with a high-pass cutoff at 10 Hz following Giandolini et al. (2016) and a low-pass cutoff at 60 Hz following Hennig and Lafortune (1991) and Gruber et al. (2014). A subsample of data was taken within a 5-s window midway through each 30-s trial representing approximately 10 and 15 steps during walking and running trials, respectively. Data from the subsample were mean-centered and de-trended, and the power of the low- and mid-back accelerometer signals during stance phase was calculated via power spectral density (PSD) analysis using Fast Fourier transformation (Hamill et al., 1995; Derrick et al., 1998; Mercer et al., 2002; Edwards et al., 2012; Gruber et al., 2014; Giandolini et al., 2012). Following Gruber et al. (2014), PSDs were computed from 0 to the Nyquist frequency and normalized to 1 Hz bins. The sum of the powers from 0 to Nyquist was used to normalize signals to their mean squared amplitudes (Gruber et al., 2014). PSD was calculated for the low- (PSD_{low}) and mid-back (PSD_{mid}) accelerations, and attenuation (or gain) in signal power between the sensors was measured using a transfer function given in decibels as:

Equation 2
$$Shock\ Attenuation(dB) = 10 \log_{10} \left(\frac{PSD_{mid}}{PSD_{low}} \right)$$

Note that negative values represent signal attenuation and positive values represent gain. This procedure was used to calculate peak power ($PP_{low,i}$, $PP_{mid,i}$) from PSD profiles and mean shock

attenuation (MSA_i), where i represents the vertical (Z), transverse (Y), and resultant (r) dimensions of accelerometer signals.

Kinematics

Three-dimensional kinematics were captured at 200 Hz using an 8-camera infrared motion capture system (Oqus 1 Series, Gothenburg, Sweden) and Qualysis tracking software (v.2.10). Small reflective markers were affixed to the 7 spinous processes approximating the T12 through S1 vertebral levels. For visualization of whole-body movements, markers were also affixed to the left and right calcaneal tuberosities, first and fifth metatarsal heads, medial and lateral maleoli, medial and lateral femoral epicondyles, greater trochanters, anterior and posterior superior iliac spines, iliac crests, acromion processes, medial and lateral humeral epicondyles, ulnar and radial styloid processes, the sternal notch, the spinous process of C7, and the frontal eminences of the forehead.

Lordosis was quantified as the Central Angle (CA) of a circle by calculating the ratio of the curved arc length of the lumbar spine divided by its radius of curvature. This method has been shown to be a reliable way to measure lumbar curvature, which correlates strongly with radiological measurements of lordosis, including Cobb angle, and allows for dynamic measures of lordosis during *in vivo* experimental studies (for discussion, see Castillo et al., 2017). Standing lordosis (Standing CA_{mean}) was computed for the best-fit, least-squares circle that passes through the 7 points representing markers on the T12-S1 spinous processes averaged over the 2-min static standing trial. To measure lordosis dynamically (Dynamic CA), the same procedure was accomplished by fitting a best-fit circle through the 7 lumbar spinous process markers for each kinematic frame of the trial. The average measurement of lordosis over the 30-s trial was used to quantify Dynamic CA_{mean} . Because lordosis oscillations were approximately sinusoidal, the average amplitude of lordosis angular displacement (CA_{amp}) was found by calculating the root mean square amplitude of Dynamic CA for an entire trial. The average velocity of

lordosis angular displacement (CA_{vel}) was found by calculating the average first order derivative of change in Dynamic CA with respect to time.

Hypothesis Testing

To test **H1**, that variations in lordosis influence lumbar shock attenuation, an ordinary least-squares (OLS) regression between lordosis and shock attenuation was conducted for walking and running. Both static standing lordosis (Standing CA_{mean}) and mean dynamic lordosis (Dynamic CA_{mean}) were tested in separate OLS regressions. The dependent variable in these models was resultant mean shock attenuation (MSA_r), which has been shown to provide a more effective measure of shock accelerations in the sagittal plane compared to analyses of individual vertical or transverse components (see Giandolini et al., 2016). Since negative values for MSA_r indicate attenuation, a negative correlation was predicted between lordosis and MSA_r to test whether more curved lumbar spines have greater capacity for shock attenuation.

The second hypothesis (**H2**), that the lumbar spine exhibits viscoelastic properties to explain impact shock attenuation during locomotion, was tested only for running because no correlation was found between walking MSA_r and lumbar lordosis. Dynamic flexion and extension of the lumbar spinal column during locomotion was assumed to behave like a simple Euler-Bernoulli beam containing both viscous and elastic elements. Dynamic changes in the beam's curvature during locomotion were assumed to be equivalent to changes in lordosis. The dynamic behavior of the beam in response to bending torques (τ) generated at impact was modeled as a function of lordosis angular displacements (θ) with respect time (t):

Equation 3
$$\tau(\theta(t)) = I \frac{d^2\theta}{dt^2} - k\theta - \nu \frac{d\theta}{dt}$$

where I represents the moment of inertia, k represents the bending stiffness coefficient of the beam, and v represents the damping coefficient of the beam. Holding constant variables I , k , and v within an individual, bending torques caused by lordosis angular accelerations are resisted by the amplitude of lordotic angular displacements and the rate of lordosis angular displacement velocity. Thus, for a given shock acceleration (holding the angular acceleration term constant), this model predicts that the stiffness and damping components should balance one another such that if angular displacements increase, then the rate of angular displacement should decrease. To test whether the lumbar spine exhibits such viscoelastic properties, the spine's elastic response to impact shocks was assumed to be inversely proportional to the amplitude of lordosis angular displacement (CA_{amp}) while its damping response was predicted to be proportional to the rate of change in lordosis angular displacement (CA_{vel}). Thus, **H2** predicts that increased shock attenuation is associated with higher CA_{amp} and lower CA_{vel} . To account for potential covariation between CA_{amp} and CA_{vel} , a multiple regression tested the effects of CA_{vel} and CA_{amp} as independent variables and running MSA_r as the dependent variable. Because the shape of the lumbar spine may also covary with viscoelastic properties, Dynamic CA_{mean} was included in the model as an additional covariate.

To further examine the relationship between lordosis and shock attenuation, **H3** tested the extent to which intervertebral discs versus standing lumbar posture explain variations in shock attenuation. An OLS regression between $relMDH$ and running MSA_r was used to test the bivariate association between disc height and lumbar shocks. To test whether lordosis is a predictor of lumbar shock attenuation after controlling for covariation with disc height, a multiple regression was used to predict running MSA_r as the dependent variable with $relMDH$ and Standing CA_{mean} as independent variables.

Statistical analysis

All processing and analyses were performed using R statistical software (version 3.1.1). Assumptions of normality in the distributions of variables were examined using a Shapiro–Wilk test. Standing CA_{mean} and Dynamic CA_{mean} were found to be log-normally distributed and therefore were log-transformed. Paired t-tests were used to compare variables between walking and running. The strength of bivariate associations was measured using Pearson’s correlations. Coefficients in the multiple regression models from **H2** and **H3** are also reported as standardized betas (β) to compare effect sizes.

Results

Table 4.1: Summary of participant anthropometrics

Variable	Mean (sd)
Height (m)	1.72 (0.09)
Body Mass (kg)	65.45 (11.45)
Standing LA_{mean} (deg)	40.5 (15.9)
MDH (cm)	9.3 (0.8)
relMDH (cm^3/kg)	12.8 (4.2)

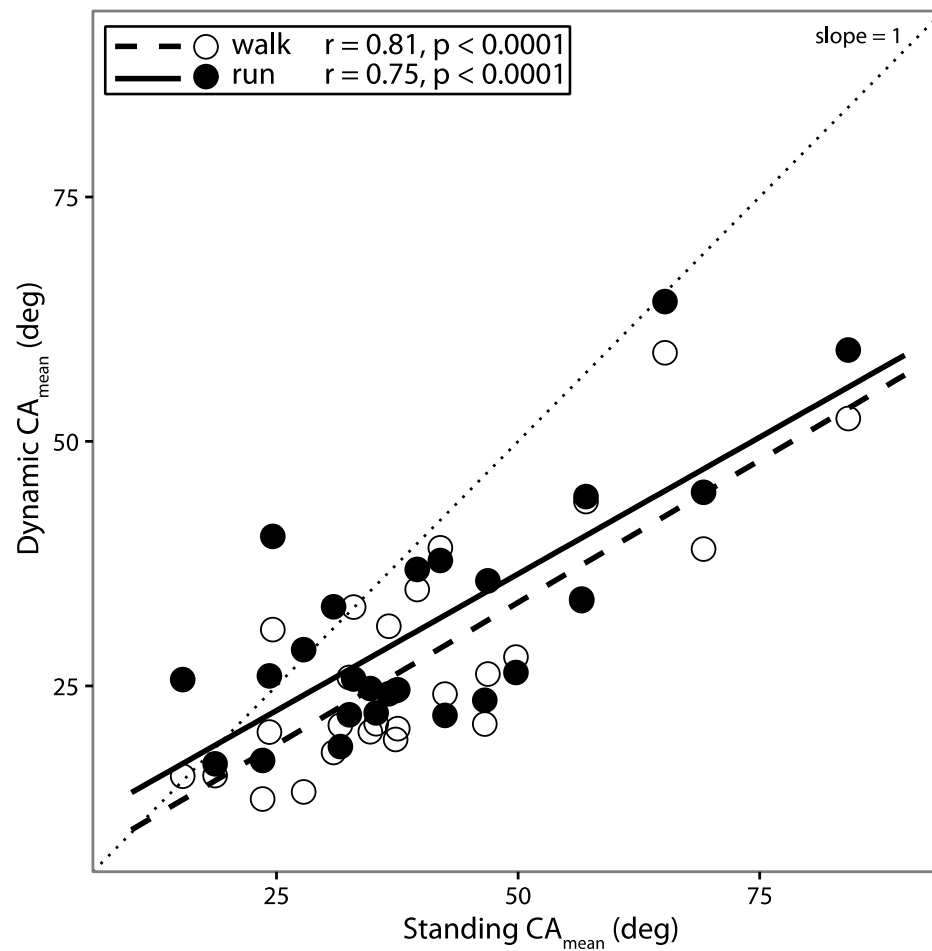


Figure 4.1: The association between static standing lordosis and dynamic lordosis during walking (open circles) and running (closed circles). There was a strong correlation between standing and dynamic postures. The regression equation for walking (dashed line) was: Dynamic $CA_{mean} = 0.58$ Standing $CA_{mean} + 4.48$. The regression equation for running (solid line) was: Dynamic $CA_{mean} = 0.56$ Standing $CA_{mean} + 8.51$.

Lordosis

Standing CA_{mean} and Dynamic CA_{mean} were strongly correlated during walking ($r = 0.81$, $p < 0.0001$) and running ($r = 0.75$, $p < 0.0001$), but the slope of the regression was 0.58 for walking and 0.56 for running, indicating that participants used straighter dynamic lumbar postures than standing postures (Figure 4.1). This effect was most pronounced among individuals with Standing $CA_{\text{mean}} > 30^\circ$. The average Standing CA_{mean} ($40.5 \pm 15.9^\circ$) was 46% greater than walking Dynamic CA_{mean} ($p < 0.0001$) and 31% greater than running Dynamic CA_{mean} ($p = 0.0002$). Comparing lordosis between gaits, Dynamic CA_{mean} was 11% greater during running than walking ($p = 0.01$, Table 4.2).

Table 4.2: Summary and comparison of dynamic variables for walking versus running.

	Walking	Running	Walking vs. running
	Mean (sd)	Mean (sd)	P-value
Dynamic Central Angle, CA_{mean} (deg)	27.8 (12.0)	30.9 (11.6)	0.01
Amplitude of lordosis angular displacement, CA_{amp} (deg)	5.5 (2.3)	7.9 (1.5)	0.0001
Velocity of lordosis angular displacement, CA_{vel} (deg/s)	42.5 (31.1)	66.8 (25.4)	0.0005
Axial mean shock attenuation, MSA_z (dB)	-1.89 (2.54)	-1.92 (1.55)	0.95
Transverse mean shock attenuation, MSA_y (dB)	0.80 (2.11)	-0.02 (4.39)	0.38
Resultant mean shock attenuation, MSA_r (dB)	2.10 (2.84)	-0.77 (3.07)	0.003
Low-back axial peak power, $PP_{\text{low},z}$ (g^2/Hz)	0.000005 (0.000004)	0.00163 (0.00056)	<0.0001
Low-back transverse peak power, $PP_{\text{low},y}$ (g^2/Hz)	0.000005 (0.000002)	0.00006 (0.00005)	<0.0001
Low-back resultant peak power, $PP_{\text{low},r}$ (g^2/Hz)	0.000605 (0.000385)	0.06480 (0.06025)	<0.0001
Mid-back axial peak power, $PP_{\text{mid},z}$ (g^2/Hz)	0.000003 (0.000002)	0.00099 (0.00030)	<0.0001
Mid-back transverse peak power, $PP_{\text{mid},y}$ (g^2/Hz)	0.000006 (0.000003)	0.00006 (0.00007)	0.0005
Mid-back resultant peak power, $PP_{\text{mid},r}$ (g^2/Hz)	0.000883 (0.000633)	0.04414 (0.03849)	<0.0001

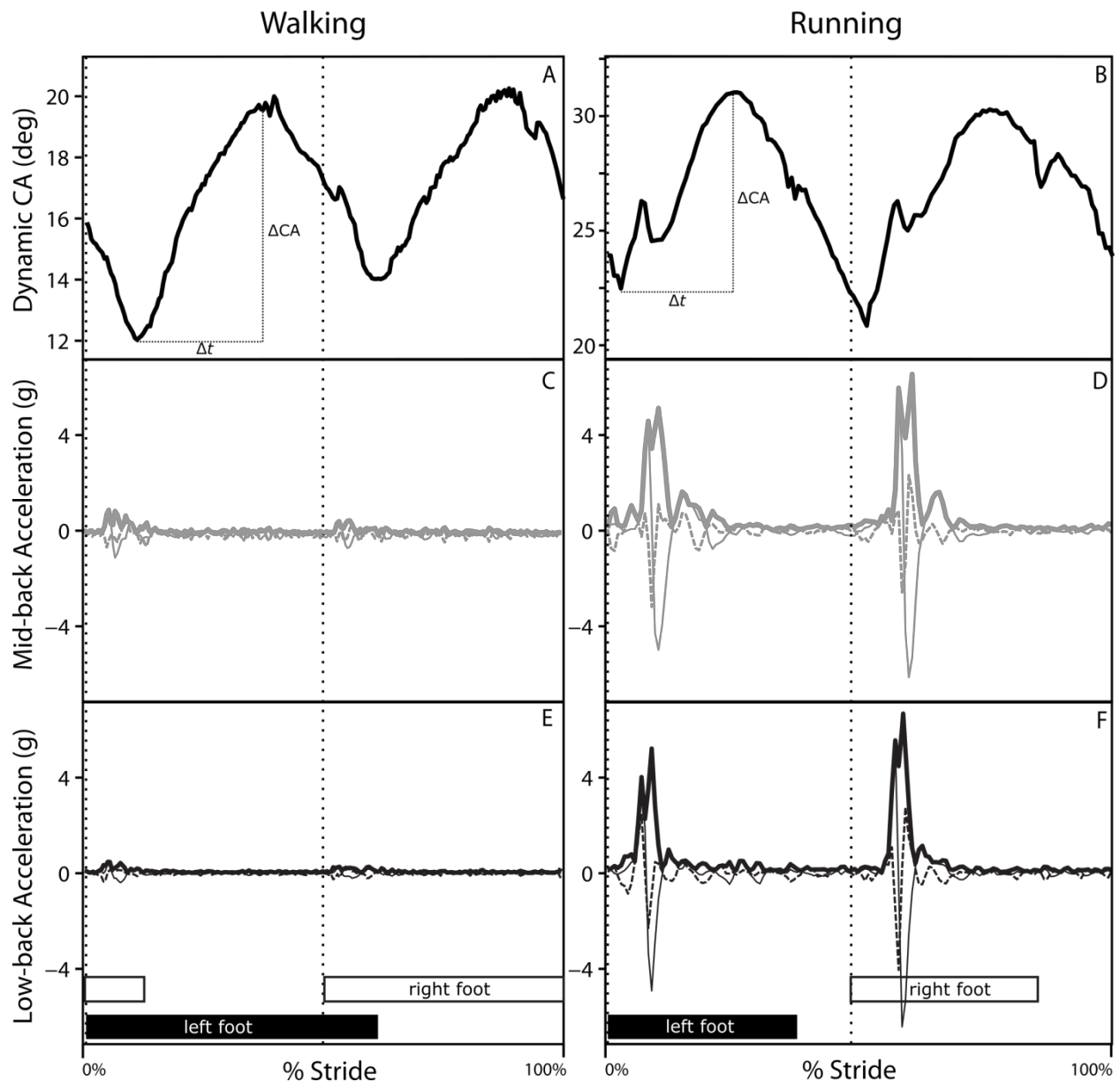


Figure 4.2: Dynamic changes in lordosis (A-B) and filtered acceleration signals in the time domain for the mid- (C-D) and low-back (E-F) during walking and running from one participant. Time was standardized to percent stride to compare between gaits. Stance time for the left (black rectangle) and right feet (open rectangle) are shown in panels E and F, with the time of foot contact illustrated as a vertical dashed line for all panels within the same gait. (A-B) Lordosis showed repeating patterns of oscillation in decreasing CA and increasing CA with each step. Changes in lordosis during spinal oscillations (ΔCA) were used to quantify the amplitude of lordosis angular displacement (CA_{amp}), and lordosis change with respect to time ($\Delta CA/\Delta t$) measured the angular velocity of lordosis displacement (CA_{vel}). (C-E) Mid- (gray lines) and low-back accelerations (black lines) are shown for the vertical (thin black line), transverse (dotted line), and resultant dimensions (thick black line).

During each step in a walking or running gait cycle, Dynamic CA showed a repeating pattern of oscillation (Figure 4.2A-B). CA_{amp} was 44% greater during running than walking ($p = 0.0001$), and CA_{vel} during running was 57% greater than walking ($p = 0.0005$, Table 4.2). There were also qualitative differences in the timing of spinal oscillation between gaits. During walking, the lumbar spine decreased in lordosis during the first third of stance phase, increased in lordosis in the middle third of stance to reach peak lordosis, and then decreased in lordosis during the last third of stance before reaching minimum lordosis at the end of double-support phase (Figure 4.2A). During running, the change from decreasing lordosis to increasing lordosis occurred immediately following impact, and the first two-thirds of stance phase were primarily associated with increasing lordosis, while the last third of stance and aerial phase showed decreasing lordosis (Figure 4.2B).

Accelerometers

The power spectra of low- and mid-back accelerometer signals showed a clear separation between mid-frequency impact ranges and high-frequency resonance ranges (Figure 4.3). The middle impact frequency component ranged from 16-27 Hz with a peak at about 23 Hz for walking (Figure 4.3A) and between 10-20 Hz with a peak at 16 Hz for running (Figure 4.3B). The higher resonance frequency component was similar across gaits, ranging between 25-35Hz with peaks at 32 Hz for walking and 30 Hz for running. Comparing between gaits, mean resultant peak power during running was about 50 times greater at the mid-back and more than 100 times greater at the low-back compared to walking ($p < 0.0001$, Table 4.2). Within gaits, mean resultant peak power was 46% greater at the mid- versus low-back accelerometer during walking ($p = 0.03$), but the opposite pattern occurred during running with peak power at the low-back 47% greater than the mid-back accelerometer ($p = 0.02$). Differences in resultant peak power were primarily driven by differences in transverse power during walking and vertical power during running. Walking transverse peak power was twice as high as vertical power at the

mid-back ($p < 0.0001$), but there were no differences between axes at the low-back. In contrast, there were no differences in transverse peak power at either the mid- or low-back during running, but running vertical peak power was 28 times larger than transverse peak power at the low-back ($p < 0.0001$) and 16.5 times larger than transverse power at the mid-back ($p < 0.0001$, Table 4.2).

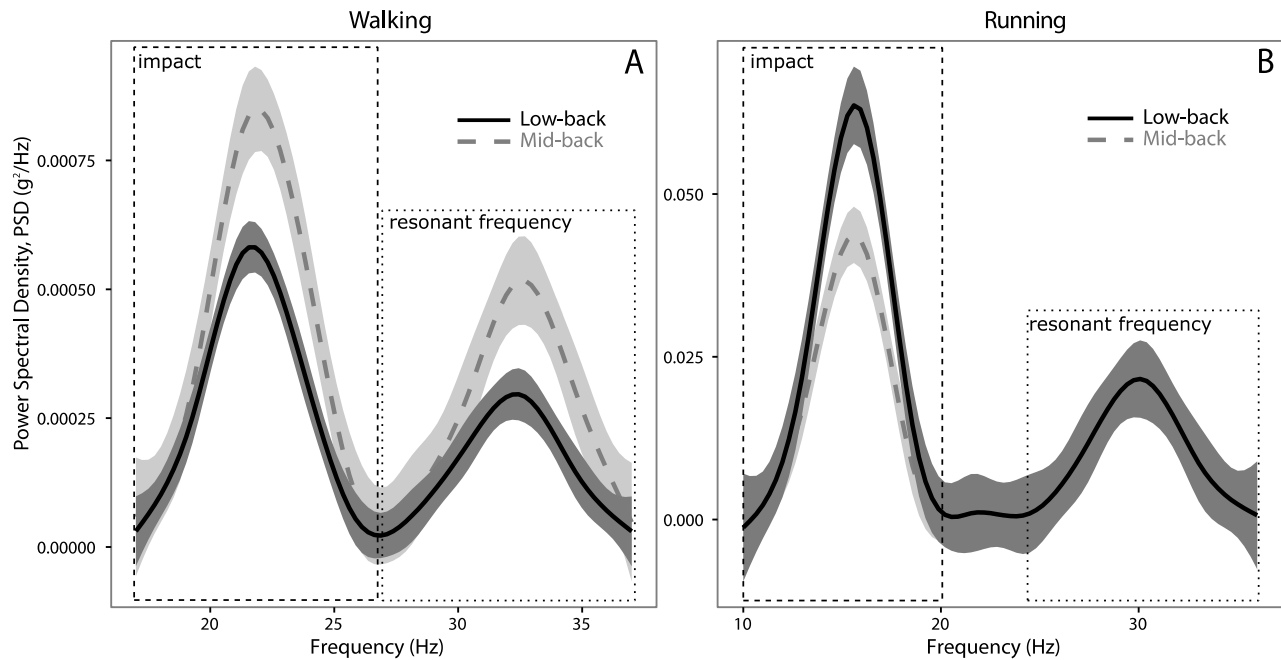


Figure 4.3: Mean power spectra for the resultant low- (black solid line) and mid-back (grey dotted line) acceleration signals across subjects, with 95% confidence intervals shown as shaded regions. Average number of steps was approximately 10 for walking and 15 for running gaits. Boxes indicate the impact and resonant frequency ranges. The impact frequency range was between 16-27 Hz with a peak at about 23 Hz for walking, and between 10-20 Hz with a peak at 16 Hz for running. The resonance frequency range for both gaits was between about 25-35 Hz with a mean at 32 Hz for walking and 30 Hz for running. Note the y-axis scale of plots is not the same for panels A and B: walking peak powers were smaller than running peak powers by several orders of magnitude.

Mean resultant shock attenuation (MSA_r) was -0.77 dB during running compared to 2.10 dB during walking ($p = 0.003$), but there were no differences between gaits for individual components of transverse or vertical shock attenuation (Table 4.2). Testing **H1**, there was no relationship between lumbar lordosis and shock attenuation during walking (Figure 4.4). Walking MSA_r was uncorrelated with Standing CA_{mean} ($r = 0.25$, $p = 0.21$) and Dynamic CA_{mean} ($r = 0.15$, $p = 0.47$). However, running MSA_r

showed a strong negative correlation with both Standing CA_{mean} ($r = -0.55$; $p = 0.004$) and Dynamic CA_{mean} ($r = -0.50$; $p = 0.009$). Since negative MSA_r values indicate attenuation, OLS regressions demonstrated that a 1% increase in standing lordosis was associated with a 9.8% increase in shock attenuation, and a 1% increase in dynamic lordosis was associated with a 10% increase in attenuation. Translating these effects from decibels to signal power ratios, running results suggest a 64% predicted reduction in low- versus mid-back signal power for the participants with the highest Dynamic CA_{mean} sampled in this study. For participants with the lowest Dynamic CA_{mean} sampled in this study, the OLS regression predicted a 42% gain in signal power between low- and mid-back during running. Also, when included as a covariate, sex had no effect on the relationship between running MSA_r and lordosis ($p = 0.09$) (Appendix Table 1.1; Appendix Figure 1.3).

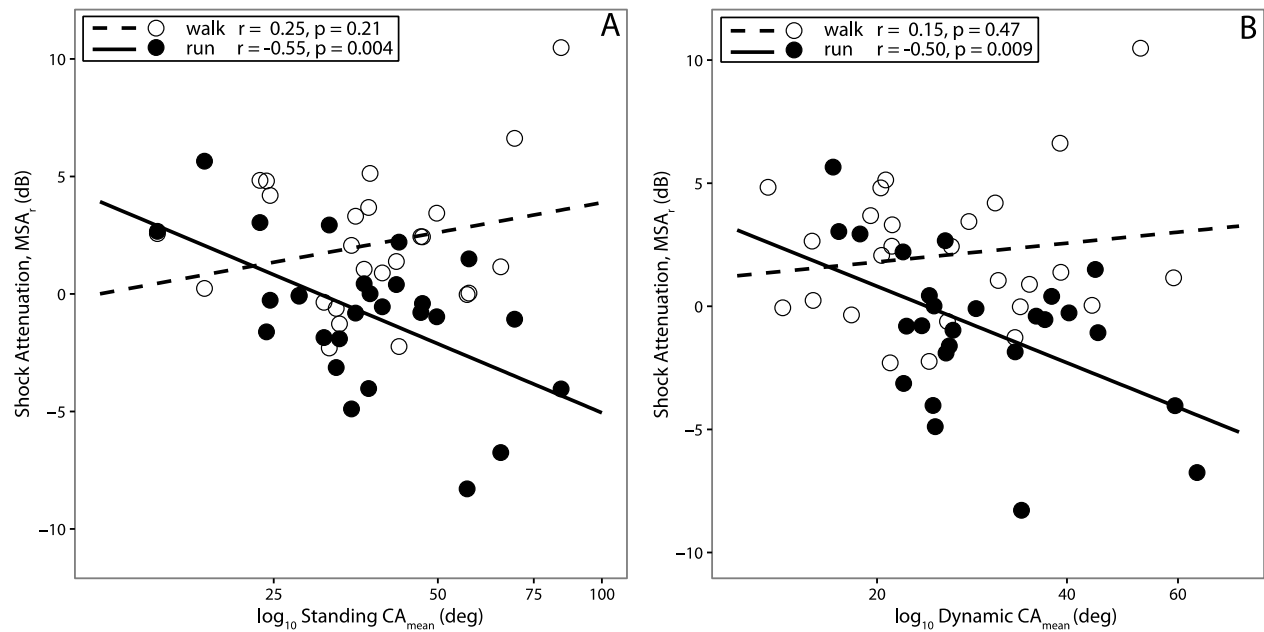


Figure 4.4: Resultant mean shock attenuation during running compared to mean static standing (A) and dynamic (B) lordosis. Attenuation was measured in decibels, so positive values for MSA_r indicate gain in signal power, negative values indicate signal attenuation, and the zero value indicates equal powers of the mid- and low-back accelerometer signals. There was no association between shock attenuation and either measure of lordosis during walking, but a strong negative relationship during running.

The amplitude of lordosis angular displacement (CA_{amp}) and lordosis angular displacement velocity (CA_{vel}) had equal but opposite effects on mean shock attenuation during running (Figure 4.5, Table 4.3). The multiple regression model revealed that CA_{amp} had a negative association with running MSA_r ($\beta = -0.40$, $p = 0.05$), suggesting that lumbar spines showing larger lordosis angular displacements had greater capacity for lumbar shock attenuation. The model also revealed that CA_{vel} had a positive association with running MSA_r ($\beta = 0.40$, $p = 0.05$), suggesting that lumbar spines with faster rates of lordosis angular displacement showed reduced capacity for lumbar shock attenuation. The model covariate, Dynamic CA_{mean_r} , was also a strong predictor of running MSA_r ($\beta = -0.53$, $p = 0.05$), indicating that lumbar curvature remained strongly associated with greater shock attenuation after controlling for the effects of viscoelasticity. The viscoelastic model explained 41% of the total variation in lumbar shock attenuation (Table 4.3).

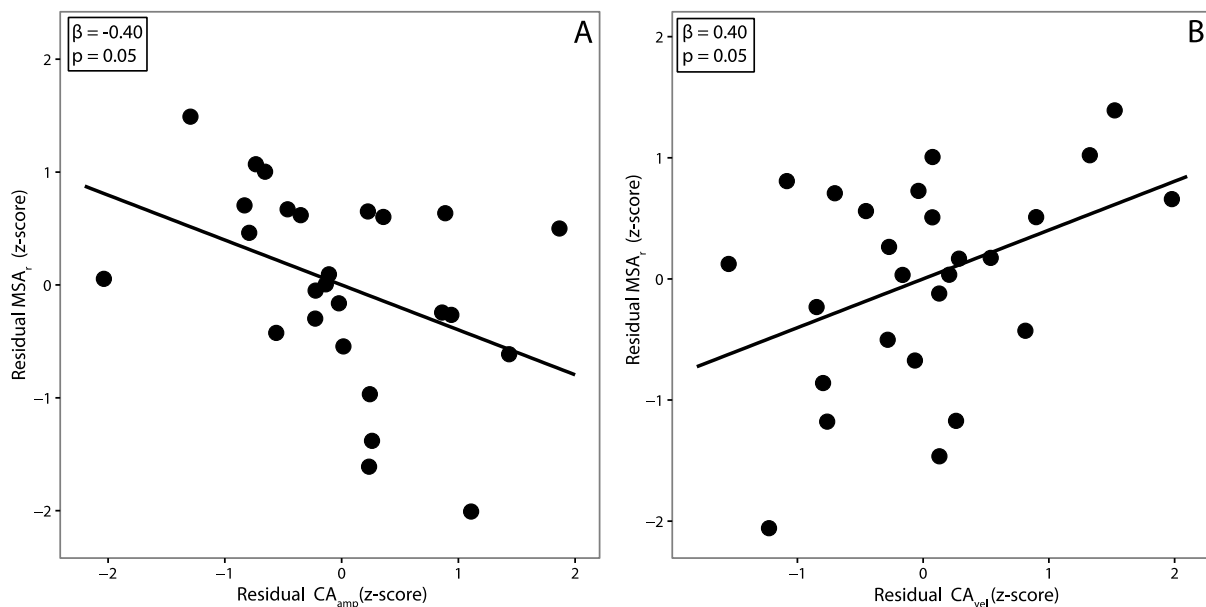


Figure 4.5: Partial regression plots showing the results from H2 testing the effects of (A) lordosis angular displacement amplitude (CA_{amp}) and (B) lordosis angular displacement velocity (CA_{vel}) on resultant mean shock attenuation (MSA_r) during running. To compare the strengths of effect sizes, variables are shown as z-scores and regressions coefficients (Table 4.3) are shown as standardized β s.

Table 4.3: Results of the multiple regression model testing **H2**, that shock attenuation is explained by the lumbar spine's viscoelastic behavior.

Variable	Coef.	β	S.E.	P-value
(Intercept)	18.07	-	5.34	0.003
CA _{amp}	-0.79	-0.40	0.38	0.05
CA _{vel}	1.95	0.40	0.94	0.05
log ₁₀ Dynamic CA _{mean}	-10.92	-0.53	3.48	0.005
Multiple R ² =0.41				

Disc height (relMDH) was strongly associated with shock attenuation capacity during running (Figure 4.6). relMDH showed a negative correlation with MSA_r ($r = -0.45$, $p = 0.02$), indicating that thicker discs attenuated greater amounts of impact shock (Figure 4.6A). Controlling for the effects of lordosis, relMDH had a negative effect on MSA_r ($\beta = -0.38$, $p = 0.02$; Figure 4.6B), but Standing CA_{mean} had an even stronger negative association with MSA_r ($\beta = -0.49$, $p = 0.005$; Figure 4.6C) (Table 4.4). These results suggest that standing lordosis was a stronger predictor of shock attenuation than intervertebral disc height.

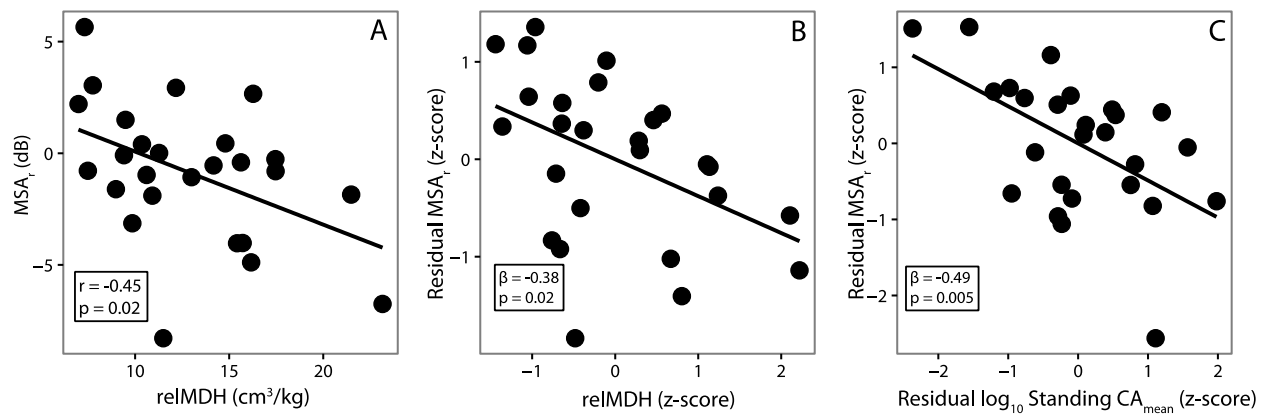


Figure 4.6: (A) The OLS regression between size-standardized max disc height (relMDH) and resultant mean shock attenuation (MSA_r) during running. (B-C) Partial regression plots showing **H3**, which tests the effects of relMDH and lordosis (Standing CA_{mean}) as predictors of MSA_r. To compare the strengths of effect sizes, variables are shown as z-scores and multiple regressions coefficients are shown as standardized β s in panels B and C.

Table 4.4: Results of the multiple regression model testing **H3**, that lumbar lordosis remains a strong predictor of lumbar shock attenuation after controlling for disc height.

Variable	Coef.	β	S.E.	P-value
(Intercept)	16.42	-	4.48	0.001
relMDH	-0.28	-0.38	0.11	0.02
$\log_{10}$ Standing LA	-8.75	0.49	2.82	0.005
Multiple $R^2 = 0.44$				

Discussion

The purpose of this study was to examine how variations in lordosis affect shock attenuation through the human lumbar spine during locomotion. Lordosis was quantified in natural standing posture and dynamically during barefoot walking and running. Although few studies have reported comparable data for dynamic flexion and extension of the lumbar spine, we found a mostly double-peaked pattern of change in lordosis over the gait cycle and 5.5° mean amplitude of total lordosis angular displacement during walking, which is consistent with the sum of segmental spinal displacements along the lumbar spine found in previous studies (Syczewska et al., 1999; Levine et al., 2007). Furthermore, this study corroborates previous work showing that running involves greater amplitudes of lordosis angular displacement and higher rates of displacement compared to walking, and that individuals use straighter lumbar postures during locomotion compared to natural standing posture (Syczewska et al., 1999; Levine et al., 2007; Dimitriadis et al., 2011).

Testing hypotheses in this study required reliable measurements of shock attenuation in the lumbar spine. We examined accelerations within the frequency domain using power spectral density analysis, which revealed distinct power peaks centered at about 23 Hz for walking and 16 Hz for running (Figure 4.3), both within the ranges of impact frequencies reported previously (James et al., 2014; Bobbert et al., 1991; Shorten and Winslow, 1992; Derrick et al., 1998; Mercer et al., 2002; Hamill et al.,

1995; Edwards et al., 2012; Gruber et al., 2014; Giandolini et al., 2016). Furthermore, we found the resonance frequency of the inertial sensor to be centered at approximately 31 Hz for both walking and running, close to the mean natural vibrating frequency of 30 Hz reported by Kitazaki and Griffin (1995), who similarly used low-mass accelerometers attached to the skin overlying lumbar spinous processes.

The first hypothesis (**H1**) tested whether increased lordosis is associated with greater lumbar shock attenuation. We found no association between lordosis and attenuation during walking but a strong association during running, explaining approximately 30% of the variation in resultant shock attenuation. For every 1% increase in lordosis there is a 10% increase in attenuation, thus strongly supporting the hypothesis that variations in lumbar posture differentially affect impact-related shock transmission through the vertebral column. Attenuation was most evident among study participants with the highest levels of lordosis, who showed a 64% predicted reduction in shock signal power transmitted through the lumbar spine. That said, resultant shock attenuation within the lumbar spine (-0.8 ± 3.1 dB) was much less intense than resultant shock attenuation measured between the tibia and sacrum (-4.0 ± 3.1 dB) (Giandolini et al., 2016). Our finding of lower levels of attenuation in the lumbar spine is likely explained by the fact that most passive and active mechanisms for attenuation take place in the lower limb, so the magnitude of shock is likely already strongly attenuated by the time it reaches the vertebral column (Paul et al., 1978; Whittle, 1999; Derrick et al., 1998; Butler et al., 2003; Edwards et al., 2012; James et al., 2014; Addison and Lieberman, 2015). Lower limb attenuation may also explain differences in lumbar shock accelerations between walking and running. Because resultant peak power at the low-back accelerometer during walking was two orders of magnitude smaller than during running (Table 4.2), it is likely that most of the impact forces generated during walking are already attenuated in the lower limb before reaching the spine. Thus, there is less of a need to attenuate shocks during walking compared to running, perhaps explaining the lack of correlation between lordosis and walking resultant shock attenuation.

The second hypothesis (**H2**) predicted that the lumbar spine exhibits viscoelastic properties to attenuate impact-related shocks. We found strong support for **H2**, which explained 41% of the variation in shock attenuation during running. Dynamic motions of the spine showed evidence that stiffer spines with less dynamic compliance also have reduced capacity for shock attenuation. Damping behavior was also observed from the association between shock attenuation and lower rates of lordosis angular displacement. Thus, lower back stiffness and damping properties may covary with lordosis to modulate lumbar shock attenuation. However, which tissues are involved in damping and how they respond to lumbar shocks remain unknown. There are numerous elastic (e.g. tendons) and viscous (e.g. discs) structures in the lumbar spine that can passively absorb or dissipate shocks, but the capacity for spinal tissues to store large amounts of strain energy and dissipate heat effectively in structures like discs (which are mostly avascular) is likely minimal (Alexander, 1997; Adams et al., 2006). Thus, we speculate that trunk muscle activity may serve as the primary factor influencing dynamic lumbar stiffness and damping behavior. More research is needed to study how muscle activation affects dynamic lordosis and lumbar shock attenuation during gait.

The third hypothesis (**H3**) examined the effects of intervertebral discs on lumbar shock attenuations. We found that the relative thickness of discs explained only 20% of the variation in resultant shock attenuation during running (Figure 4.6). But after controlling for the effects of lordosis, lumbar curvature had an even stronger influence on shock attenuation than intervertebral discs, which together explained more than twice the variation (44%) in lumbar shock attenuation compared to disc height alone. Intervertebral discs, which account for the majority of variation in lumbar curvature in adults (Shefi et al., 2013), are often viewed as the major “shock absorbers” of the lumbar spine, but these findings suggest that sagittal spinal shape may be a more important factor in attenuating impact accelerations.

This study further highlights the importance of analyzing both transverse and vertical components of impact shock during locomotion. As previous authors have concluded, transverse and resultant accelerations provide crucial data for shock assessment compared to analyses of vertical accelerations alone (Giandolini et al., 2016). For example, the dimensional contributions of peak power explained differences in shock attenuation between gaits and the relationship between shock attenuation and lordosis. The main difference between walking and running accelerations was elevated transverse peak power during walking. Walking transverse peak power was twice as high as vertical power at the mid-back, causing an average gain in signal rather than attenuation—which is not unusual in studies of impact shock and most commonly attributed to body segment motion, oscillations of the center of mass, vibrations near resonance frequency, or joint kinematics during the stance phase (Shorten and Winslow; 1992; Hammill et al., 1995; Gruber et al., 2014). Our finding of high transverse shock accelerations during walking may be due to overall greater antero-posterior ground reaction impulse forces reported for barefoot walking. Nilsson and Thorstensson (1989) examined ground reaction forces according to foot strike patterns and showed that individuals walking barefoot at 1.5 m/s had antero-posterior braking impulse forces that were roughly two-thirds greater than individuals using a rearfoot strike pattern while running at 3 m/s. Since previous studies of shock attenuation during barefoot walking have only considered vertical shock accelerations (e.g. James et al., 2014), it is unknown whether elevated transverse shocks found in this study are representative of barefoot walking overall.

During running, peak power in the vertical axis was 16 to 28 times the peak power in the transverse axis. This finding suggests that the correlation between lordosis and shock attenuation is primarily driven by vertical rather than transverse forces. Though the mechanisms underlying dimensional differences in running acceleration peaks and their relation to lumbar spinal shape are still unclear, one explanation may be that vertical peak power is related to gait kinematics. Greater pelvic tilt

(and presumably higher lordosis) is known to be associated with longer strides and stiffer lower limb postures (Vialle et al., 2005; Franz et al., 2009; Hamill et al., 2009). Thus, it is possible that increased lower extremity stiffness causes higher levels of vertical impact forces during running. However, future work is necessary to better understand how lumbopelvic motions interact and to what extent they influence shock attenuation.

There were several limitations in this study. First, only healthy young adults (mostly students) were recruited, thus sampling only a narrow range of variation in physical activity levels, age, and other factors that may influence lumbar shock attenuation. Second, this study relied on external measures of lordosis. Although the central angle method used here strongly correlates with radiological standards (e.g. Cobb LA), this method is likely less precise than other radiological imaging methods (see Castillo et al., 2017). Third, these experiments were conducted on barefoot participants, and although participants were instructed to use a rearfoot strike pattern during all trials, we did not measure or control for foot strike pattern. There is an extensive literature demonstrating the effects of footwear and strike pattern on impacts during locomotion, which could have influenced the results in this study (Boyer et al., 2014; Divert et al., 2005; Lieberman et al., 2010; Kulmala et al., 2013; Giandolini et al., 2016). However, a post-hoc qualitative examination of the force plate data collected in this study found that 25 out of 27 participants had clearly visible and consistent impact transients in their vertical ground reaction forces. Since forefoot strike patterns are associated with reduced impact transients (Lieberman et al., 2010), most of the subjects in this study are assumed to have used rearfoot strikes, so it is unlikely that foot strike had a strong effect on the relationship between lordosis and lumbar shock attenuation measured in this study. Fourth, we analyzed only one speed for each gait. Though speed may influence shock attenuation, our goal was to study only moderate, comfortable gait speeds during which we could establish baseline measures of lumbar shock attenuation and spinal motion. Other studies might consider examining the relationship between lordosis and lumbar shock acceleration across speeds.

Finally, this study did not examine other features of active shock attenuation such as lower limb kinematics or trunk muscle activation. Because the focus of this experiment was to understand the relationship between lumbar posture (and changes in posture) on lumbar shocks, this study only examined variables related to spinal motion and spinal accelerations. However, future work would greatly benefit from testing how lower limb joint stiffness or trunk muscle activation may affect the patterns of shock attenuation found here.

In conclusion, the key finding of this study is that higher amounts of lumbar curvature reduce impact-related shocks during running but not walking. This result strongly supports the hypothesis that lumbar spinal shape is particularly important during dynamic activities. Not only does this have evolutionary implications for interpreting lordosis variations in fossil hominins, but the relationship between shock attenuation and lordosis may explain the pattern of lumbar curvature observed among modern athletes. Evidence suggests that lordosis is higher among athletes for whom running and dynamic impacts are key (e.g. sprinters, long distance runners, and soccer players), but athletes who do not experience high levels of impact-related loading (e.g. body builders and swimmers) tend to have much straighter lumbar spines (Uetake and Ohtsuki, 1993; Wodecki et al., 2002). Though other factors may covary with lordosis differences (i.e. trunk muscle strength and flexibility; Castillo et al., 2017), this study may suggest that lordosis differences serve a functional role among modern athletes.

Given the advantages of greater lumbar curvature for shock attenuation, why are there such high levels of variability among modern humans? One hypothesis is that novel environmental conditions explain high levels of lordosis variability today. As we recently argued, reduced physical activity levels and novel behaviors since the industrial revolution—such as sleeping on soft mattresses and prolonged sitting in chairs throughout the day—may have led to abnormally low patterns of spinal loading and weaker, less stable back tissues (Castillo and Lieberman, 2015). Testing this hypothesis requires detailed

comparative studies of populations around the world that vary in activity levels and spinal loading behaviors (e.g. habitually carrying heavy loads, using harder sleeping surfaces), such as hunter-gatherers and non-industrial societies. Another hypothesis is that the evolution of human lumbar spinal curvature represents tradeoffs between competing selective pressures. In the context of this study, one important tradeoff arises between injury risk and effective shock dissipation. On the one hand, attenuation of impact-related shocks is crucial for head stabilization, preservation of vision during locomotion, and dynamic stability (Pozzo et al. 1990, 1991; Whittle, 1999). On the other hand, high levels of lordosis and shock attenuation, especially via passive means, can cause tissue strain and injury (Hamill et al., 1995). Clinical evidence suggests a link between lordosis and spinal pathology (Berlemann et al., 1999; Umehara et al.; 2000; Kumar et al., 2001; Rajnics et al., 2002; Labelle et al., 2005; Barrey et al., 2007; Chen et al., 2009). Furthermore, the viscoelastic hypothesis (**H2**) tested in this study shows that increased shock attenuation is associated with increased dynamic lordosis angular displacements, possibly suggesting higher amounts of strain in lumbar tissues. We also demonstrate that the passive effects of intervertebral discs account for about 20% of shock attenuation (**H3**), further suggesting potentially high strain on discs during dynamic changes in lumbar posture. Additionally, experimental research has shown that individuals with back pain show reduced shock attenuation capacity (Voloshin and Wosk, 1982) and a reduction in disc height following prolonged dynamic activities such as running (Dimitriadis et al., 2011). Thus, curved lumbar spines may allow for greater shock attenuation at the cost of increased risk of injury. However, straighter lumbar spines, which may be more stable and less injury prone, are less able to contend with shock forces generated during running. Understanding this tradeoff has important implications for the interpreting lordosis variability in the fossil record and for studying how dynamic impacts correlate with injury risk, which will be explored in the final chapter of this thesis.

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References

- Adams MA, Hutton WC. 1985. The effect of posture on the lumbar spine. *J Bone Joint Surg* 67(4): 625-9.
- Adams MA, Bogduk N, Burton AK, Dolan P. 2006. *The biomechanics of back pain*. Churchill Livingstone, Edinburgh.
- Addison BJ, Lieberman DE. 2015. Tradeoffs between impact loading rate, vertical impulse and effective mass for walkers and heel strike runners wearing footwear of varying stiffness. *J Biomech* 48(7): 1318-24.
- Alexander R, Jayes A. 1983. A dynamic similarity hypothesis for the gaits of quadrupedal mammals. *J Zool Lond* 201: 135–52.
- Alexander, R.M., 1997. Elasticity in human and animal backs. Vleeming A, Mooney V, Dorman T. Snijders CJ, Stoeckart R, eds. *Movement, Stability and Low Back Pain*. Edinburgh, UK: Churchill Livingstone.
- Angeloni C, Riley PO, Krebs DE. 1994. Frequency content of whole body gait kinematic data. *IEEE Trans Rehabil Eng* 2(1): 40-6.
- Been E, Gómez-Olivencia A, Kramer PA. 2012. Lumbar lordosis of extinct hominins. *Am J Phys Anthropol* 147: 64–77.
- Berlemann U, Jerszensky DJ, Bühler DW, Harms J. 1999. The role of lumbar lordosis, vertebral end-plate inclination, disc height, and facet orientation in degenerative spondylolisthesis. *J Spinal Disord* 12: 68-73.
- Bobbert MF, Schamhardt HC, Nigg BM. 1991. Calculation of vertical ground reaction force estimates during running from positional data. *J Biomech* 1991 24(12): 1095-105.
- Botsford DJ, Esses SI, Ogilvie-Harris DJ. 1994. In vivo diurnal variation in intervertebral disc volume and morphology. *Spine* 19: 935-40.
- Boyer ER1, Rooney BD, Derrick TR. 2014. Rearfoot and midfoot or forefoot impacts in habitually shod runners. *Med Sci Sports Exerc* 46(7): 1384–91
- Butler RJ, Crowell III HP, McClay Davis I. 2003. Lower extremity stiffness: implications for performance and injury. *Clin Biomech* 18: 511–7.
- Castillo ER, Hsu C, Mair RW, Lieberman DE. 2017. Testing biomechanical models of human lumbar lordosis variability. *Am J Phys Anthropol* 00: 1–12. <https://doi.org/10.1002/ajpa.23189>
- Castillo ER, Lieberman DE. 2015. Lower back pain. *Evol Med Public Health*. 1: 2-3.
- Chen IR, Wei TS. 2009. Disc height and lumbar index as independent predictors of degenerative spondylolisthesis in middle-aged women with low back pain. *Spine* 34: 1402–9.
- Davis IS, Bowser BJ, Mullineaux DR. 2016. Greater vertical impact loading in female runners with medically diagnosed injuries: a prospective investigation. *Br J Sports Med* 50: 887-92.
- Delgado TL, Kubera-Shelton E, Robb RR, Hickman R, Wallmann HW, Dufek JS. 2013. Effects of foot strike on low back posture, shock attenuation, and comfort in running. *Medicine and science in sports and exercise* 45(3):490-6.

- Derrick TR, Hamill J, Caldwell GE. 1998. Energy absorption of impacts during running at various stride lengths. *Med Sci Sports Exerc* 30:128–35
- Dimitriadis AT, Papagelopoulos PJ, Smith FW, Mavrogenis AF, Pope MH, Karantanas AH, Hadjipavlou AG, Katonis PG. 2011. Intervertebral disc changes after 1 h of running: a study on athletes. *J Int Med Res* 39(2): 569-79.
- Divert C, Mornieux G, Baur H, Mayer F, Belli A. 2005. Mechanical comparison of barefoot and shod running. *Int J Sports Med* 26(07): 593-8.
- Edwards WB, Derrick TR, Hamill J. 2012. Musculoskeletal attenuation of impact shock in response to knee angle manipulation. *J Appl Biomech* 28: 502-10.
- Forner-Cordero A, Mateu-Arce M, Forner-Cordero I, Alcántara E, Moreno JC, Pons JL. 2008. Study of the motion artefacts of skin-mounted inertial sensors under different attachment conditions. *Physiol Meas* 29(4): N21-31.
- Franz JR, Paylo KW, Dicharry J, Riley PO, Kerrigan DC. 2009. Changes in the coordination of hip and pelvis kinematics with mode of locomotion. *Gait & Posture* 29(3): 494-8.
- Giandolini M, Horvais N, Rossi J, Millet GY, Samozino P, Morin JB. 2016. Foot strike pattern differently affects the axial and transverse components of shock acceleration and attenuation in downhill trail running. *J Biomech* 49(9):1765-71.
- Gracovetsky SA, Iacono S. 1987. Energy transfers in the spinal engine. *J Biomed Eng* 9: 99–114.
- Grasso R, Zago M, Lacquaniti F. 2000. Interactions between posture and locomotion: motor patterns in humans walking with bent posture versus erect posture. *J Neurophysiol* 83: 288–300.
- Gruber AH, Boyer KA, Derrick TR, Hamill J. 2014. Impact shock frequency components and attenuation in rearfoot and forefoot running. *J Sport Health Sci* 3(2):113-21.
- Hamill J, Derrick TR, Holt KG. 1995. Shock attenuation and stride frequency during running. *Hum Mov Sci* 14: 45–60
- Hamill J, Moses M, Seay J. 2009. Lower extremity joint stiffness in runners with low back pain. *Res Sports Med* 17(4): 260-73.
- Hennig EM, Lafortune MA. 1991. Relationships between ground reaction force and tibial bone acceleration parameters. *Int J Sports Biomech* 7: 303-9.
- Herrmann L. 2008. Vibration of the Euler-Bernoulli beam with allowance for dampings. *Proceedings of the World Congress on Engineering*. Vol II WCE 2008, July 2 - 4, 2008, London.
- Hirose D, Ishida K, Nagano Y, Takahashi T, Yamamoto H. 2004. Posture of the trunk in the sagittal plane is associated with gait in community-dwelling elderly population. *Clin Biomech* 19(1): 57-63.
- James DC, Mileva KN, Cook DP. 2014. Low-frequency accelerations over-estimate impact-related shock during walking. *J Electromyogr Kinesiol* 24(2): 264-70.
- Kitazaki S, Griffin MJ. 1995. A data correction method for surface measurement of vibration on the human body. *J Biomech* 28(7): 885-90.

- Kulmala JP, Avela JA, Pasanen KA, Parkkari JA. 2013. Forefoot strikers exhibit lower running-induced knee loading than rearfoot strikers. *Med Sci Sports Exerc* 45(12) :2306-13.
- Kumar MN, Baklanov A, Chopin D. 2001. Correlation between sagittal plane changes and adjacent segment degeneration following lumbar spine fusion. *Eur Spine J* 10: 314-19.
- Labelle H, Roussouly P, Berthonnaud E, Dimnet J, O'Brien M. 2005. The importance of spino-pelvic balance in L5-s1 developmental spondylolisthesis: a review of pertinent radiologic measurements. *Spine* 30: S27–34.
- Lampl M. 1992. Further observations on diurnal variation in standing height. *Ann Hum Biol* 19: 87–90.
- Levine, D., Colston, M.A., Whittle, M.W., Pharo, E.C. and Marcellin-Little, D.J., 2007. Sagittal lumbar spine position during standing, walking, and running at various gradients. *Journal of athletic training*, 42(1), p.29.
- Lieberman DE, Venkadesan M, Werbel WA, Daoud AI, D'Andrea S, Davis IS, Mang'Eni RO, Pitsiladis Y. 2010. Foot strike patterns and collision forces in habitually barefoot versus shod runners. *Nature* 463(7280): 531-5.
- McMahon TA, Valiant G, Frederick EC. 1987. Groucho running. *J Appl Phys* 62(6): 2326-37.
- Mercer JA, Vance J, Hreljac A, Hamill J. 2002. Relationship between shock attenuation and stride length during running at different velocities. *Eur J Appl Physiol* 87(4): 403-8.
- Nilsson J, Thorstensson A. 1989. Ground reaction forces at different speeds of human walking and running. *Acta Physiol Scand* 36(2): 217-27.
- Nokes L, Fairclough J A, Mintowt-Czyz W J, Mackie I and Williams J. 1984. Vibration analysis of human tibia: the effect of soft tissue on the output from skin-mounted accelerometers *J Biomed Eng* 6: 223–6.
- Ogon M, Aleksiev AR, Spratt KF, Pope MH, and Saltzman CL. 2001. Footwear affects the behavior of low back muscles when jogging. *Int J Sports Med* 22: 414-19.
- Paul IL, Munro MB, Abernethy PJ, Simon SR, Radin EL, Rose RM. 1978. Musculo-skeletal shock absorption: relative contribution of bone and soft tissues at various frequencies. *J Biomech* 11: 237-9.
- Perl DP, Daoud AI, Lieberman DE. 2012. Effects of footwear and strike type on running economy. *Med Sci Sports Exerc* 44(7):1335-43.
- Pontzer H, Raichlen DA, Rodman PS. 14. Bipedal and quadrupedal locomotion in chimpanzees. *J Hum Evol* 66: 64-82.
- Pozzo T, Berthoz A, Lefort L, Vitte E. 1991. Head stabilization during various locomotor tasks in humans. II. Patients with bilateral peripheral vestibular deficits. *Exp Brain Res* 85(1): 208–17.
- Pozzo T, Berthoz A, Lefort L. 1990. Head stabilization during various locomotor tasks in humans. I. Normal subjects. *Exp Brain Res* 82(1): 97–106.

- Rajnice P, Templier A, Skalli W, Lavaste F, Illes T (2002) The importance of spinopelvic parameters in patients with lumbar disc lesions. *International orthopaedics* 26:104–108.
- Rak Y. 1993. Morphological variation in *Homo neanderthalensis* and *Homo sapiens* in the Levant: a biographic model. In: KimbelKimbel WH, Martin LB, editors. *Species, species concepts, and primate evolution*. New York: Plenum Press. p 523–536.
- Saha S, Lakes RS. 1977. The effect of soft-tissue on wave propagation and vibration tests for
- Sarwahi V, Boachie-Adjei O, Backus SI, Taira G. 2002. Characterization of gait function in patients with postsurgical sagittal (flatback) deformity: a prospective study of 21 patients. *Spine* 27(21): 2328-37.
- Schwab F, Lafage V, Boyce R, Skalli W, Farcy JP. 2006. Gravity Line Analysis in Adult Volunteers. *Spine* 31: E959-E967.
- Shefi S, Soudack M, Konen E, Been E. 2013. Development of the lumbar lordotic curvature in children from age 2 to 20 years. *Spine* 38(10): E602-8.
- Shorten MR, Winslow DS. 1992. Spectral analysis of impact shock during running. *Int J Sport Biomech* 8: 288–304
- Strickland AL, Shearin RB. 1972. Diurnal height variation in children. *J Pediatr* 80: 1023-1025.
- Syczewska M, Öberg T, Karlsson D. 1999. Segmental movements of the spine during treadmill walking with normal speed. *Clin Biomech* 14(6): 384-388.
- Tillmann V, Clayton PE. 2001. Diurnal variation in height and the reliability of height measurements using stretched and unstretched techniques in the evaluation of short-term growth. *Ann Hum Biol* 28: 195–206.
- Trujillo D M and Busby H R 1990 A mathematical method for the measurement of bone motion with skin-mounted accelerometers *J. Biomech. Eng.* 112 229–31
- Umehara S, Zindrick MR, Patwardhan AG, Havey RM, Vrbos LA, Knight GW, Miyano S, Kirincic M, Kaneda K, Lorenz MA. The biomechanical effect of postoperative hypolordosis in instrumented lumbar fusion on instrumented and adjacent spinal segments. *Spine*. 2000; 25, 1617–1624.
- Vialle R, Levassor N, Rillardon L, Templier A, Skalli W, Guigui P. 2005. Radiographic Analysis of the sagittal alignment and balance of the spine in asymptomatic subjects. *J Bone Joint Surg Am* 87: 260–267.
- Voloshin A, Wosk J, Brull M. 1981. Force wave transmission through the human locomotor system. *Trans ASME J Biomech Eng* 103: 48–50.
- Voloshin A, Wosk J. 1982. An in vivo study of low back pain and shock absorption in the human locomotor system. *J Biomech* 15: 21–27.
- Voloshin AS, Mizrahi J, Verbitsky O, Isakov E. 1998 Dynamic loading on the human musculoskeletal system—effect of fatigue. *Clin Biomech* 13(7):515-20.
- Voss LD, Bailey BJ. 1997. Diurnal variation in stature: is stretching the answer? *Arch Dis Child*. 77: 319–322.

- Uetake T, Ohtsuki F. 1993. Sagittal configuration of spinal curvature line in sportsmen using Moire technique. *Okajimas Folia Anat Jpn* 70:91–103.
- Wagner H, Liebetrau A, Schinowski D, Wulf T, de Lussanet MH. 2012. Spinal lordosis optimizes the requirements for a stable erect posture. *Theor Biol Med Model* 9(1): 13.
- Walker ML, Rothstein JM, Finucane SD, Lamb RL. 1987. Relationships between lumbar lordosis, pelvic tilt, and abdominal muscle performance. *Phys Ther* 67: 512–516.
- Whitehouse RH, Tanner JM, Healy MJ. 1974. Diurnal variation in stature and sitting height in 12-14-year-old boys. *Ann Hum Biol* 1: 103–106.
- Whittle MW. 1999. Generation and attenuation of transient impulsive forces beneath the foot: a review. *Gait & Posture* 10(3): 264-275.
- Wodecki P, Guigui P, Hanotel MC, Cardinne L, Deburge A. 2002. Sagittal alignment of the spine: comparison between soccer players and subjects without sports activities. *Revue de chirurgie orthopedique et reparatrice de l'appareil moteur* 88(4):328-36.
- Ziegert JC, Lewis JL. 1979. The effect of soft tissue on measurements of the vibrational bone motion by skin-mounted accelerometers. *ASME Journal of Biomechanical Engineering* 101, 218-220.

Chapter 5 – Discussion

Results presented in this thesis have important implications for human postural evolution and for understanding the etiology of modern lumbar pathology. The first section of this chapter summarizes the main results of Chapters 2 through 4; the second section considers the implications of these results for speculating about the evolutionary forces that shaped the hominin lumbar spine; and the third section discusses the origins of lordosis in relation to the rising prevalence of disorders such as back pain. This chapter concludes by recapitulating the major conclusions of the thesis and speculates about future directions for research.

Chapter Summaries

Chapter 2 relied on magnetic resonance imaging (MRI) and a 3-D postural analysis to test three biomechanical models. The first model tested the relationship between external hip flexor moments and lordosis, but the results did not support a correlation between these variables. The second model tested whether the lumbar spine behaves like a loaded bent beam and found that the beam model explained 25% of the variation in standing but not supine lordosis. Lumbar vertebral body cross-sectional area, lumbar length, and spinal flexion moments were negatively correlated with lordosis. The third model tested a tradeoff between relative trunk strength and sagittal lumbar flexibility. The strength-flexibility model was strongly supported, explaining the most variation (65%) in standing lordosis. This model found that lumbar curvature was strongly associated with relative trunk strength among individuals with high lumbar range of motion such that stronger back muscles were associated with greater lumbar curvature while stronger abdominal muscles were associated with reduced spinal curvature. However, the interaction between variables revealed that individuals with reduced lumbar range of motion also had reduced lordosis regardless of trunk strength variations. In addition, a negative association between relative back strength (controlling for abdominal strength) and trunk flexibility was found, suggesting a tradeoff between back musculature and spinal mobility.

Chapter 3 tested how variability in lordosis affects patterns of lumbar deformation and bending resistance during axial spinal loading. A weight vest experiment tested lumbar spinal deformations using loads of 0-30% body mass added in 5% increments. External measurements of lumbar posture were combined with MRI imaging and a curved beam bending model to test three main hypotheses. The first hypothesis tested whether lordosis decreases with added load to increase mechanical stability. Some support was found for a negative correlation between average lordosis and load mass. The second hypothesis tested how posture affects time-dependent patterns of lumbar deformation. Within a loading trial, results showed that relatively straighter spines were less variable and tended to straighten during loading, while curved spines tended to show more lordosis variability and become more curved during loading. The last hypothesis predicted an inverse relationship between lordosis and the spine's resistance to bending. Strong support was found for a negative correlation between unloaded lordosis and bending resistance, which explained 52% and 64% of the variation in lumbar spinal bending stiffness and elastic modulus, respectively.

Chapter 4 explored the relationship between lordosis and lumbar shock attenuation during barefoot walking and running. Three hypotheses were tested using two accelerometers mounted at the mid- and low-back. The first hypothesis was that more curved lumbar spines have greater capacity for lumbar shock attenuation. No association was found between lordosis and shock attenuation during walking, but a strong association was found during running, which explained 30% of the variation in attenuation. The second hypothesis, which tested a viscoelastic model of dynamic lumbar motion, was supported by associations between increased shock attenuation and higher degrees of dynamic lordosis angular displacements and slower rates of displacement. The viscoelastic model explained 41% of the variation in attenuation. The third hypothesis tested the effects of intervertebral disc height on shock attenuation. Although thicker intervertebral discs were found to be associated with increased shock

attenuation, lordosis was a stronger predictor of attenuation when controlling for both intervertebral discs and lumbar posture.

The evolution of hominin lumbar lordosis

Although a small degree of lordosis has been observed in other animals, including non-human primates, the pronounced curvature of the human lower back is uniquely derived (Schultz, 1961). Lordosis was a crucial adaptation for bipedalism that allowed hominins to balance the mass of the upper body over the lower limb. Although no fossil lumbar vertebrae are preserved among any of the early hominins, Lovejoy et al. (2009) have argued that the anatomy of the pelvis of *Ardipithecus ramidus* implies that lumbar curvature was present in early hominins by at least 4.4 mya. However, direct evidence of lordosis is sparse but suggests that most hominins had lumbar curvature within the modern human range with the notable exception of Neanderthals (Been et al., 2012). Comparing Cobb angles from participants in this study to fossil estimates, there is no difference between modern lordosis and early modern humans, *Homo erectus*, or australopiths (Figure 5.1A). But modern humans in this study had 60% greater lordosis than Neanderthals ($p < 0.001$) and more than double the lordosis of modern apes ($p < 0.000001$). However, this thesis primarily used the standing Central Angle (CA) method to quantify lumbar curvature because it captures more variation in lordosis than Cobb and can be more easily incorporated into static and dynamic biomechanical studies (Castillo et al., 2017). When using a regression model to predict CA from Cobb angle, there is no difference in CA among hominins, including Neanderthals who are at the low range of modern CA but not significantly different at conventional alpha levels ($p = 0.08$). Apes, however, had 57% straighter CAs than modern humans ($p < 0.01$) (Figure 5.1B).

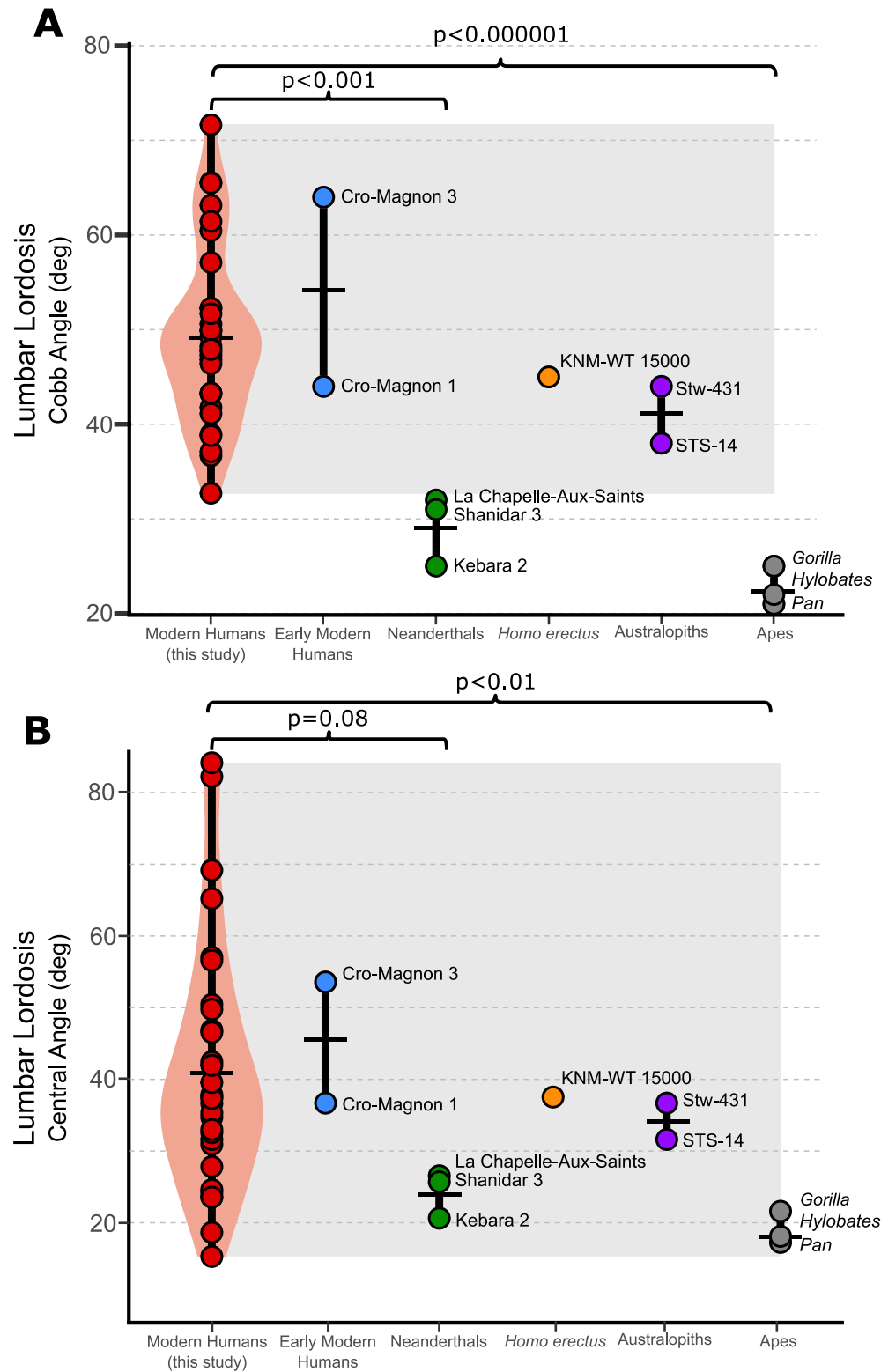


Figure 5.1: (A) Violin plots of Cobb angles measured from participants in this study (red; $n = 30$) compared to estimates of Cobb angle for various hominoids (values from Been et al., 2012). (B) Using a regression equation relating Cobb angle to the standing Central Angle used throughout this thesis, an alternative calculation of lordosis is shown for these same taxa.

Variability among fossil species presents the question of why most hominins had similar degrees of lordosis while Neanderthals had much straighter lower backs. Results presented in Chapters 2 through 4 may provide clues. In particular, one hypothesis posed in Chapter 2 is that some variability in hominin lumbar curvature was due to differences in body size and shape. The beam bending model suggests that forward flexion moments acting on the lumbar spine may have been higher in Neanderthals, who had larger upper bodies with deeper rib cages and longer pubic bones compared to modern humans (Rak and Arensburg, 1987; Franciscus and Churchill, 2002), often considered adaptations for cold environments to decrease body surface area relative to volume (Holliday, 1997). It is possible that the antero-posterior expansion of the Neanderthal trunk may have caused an anterior shift in the center of mass of their upper bodies, thereby passively increasing bending moments that act to straighten the lumbar spine. Given the fragmentary nature of fossil material, it is difficult to estimate Neanderthal lumbar bending moments based on reconstructions of the thorax. That said, Chapter 2 also revealed that lumbar vertebral body cross-sectional area had a moderate negative association with standing lumbar lordosis ($r = -0.33$). To test fossil correlates of the beam bending model, the nearly complete lumbar series from Kebara 2 may provide clues. Comparing surface area values reported in Been et al. (2010) to MRI measurements from this study, Neanderthal lumbar bodies were 25-53% larger than modern humans in this sample ($p < 0.05$) (Figure 5.2). Though more data are needed, the larger lumbar vertebral bodies of Neanderthals may support the hypothesis that reduced lordosis in part may have been due to the passive effects of upper body shape and proportions.

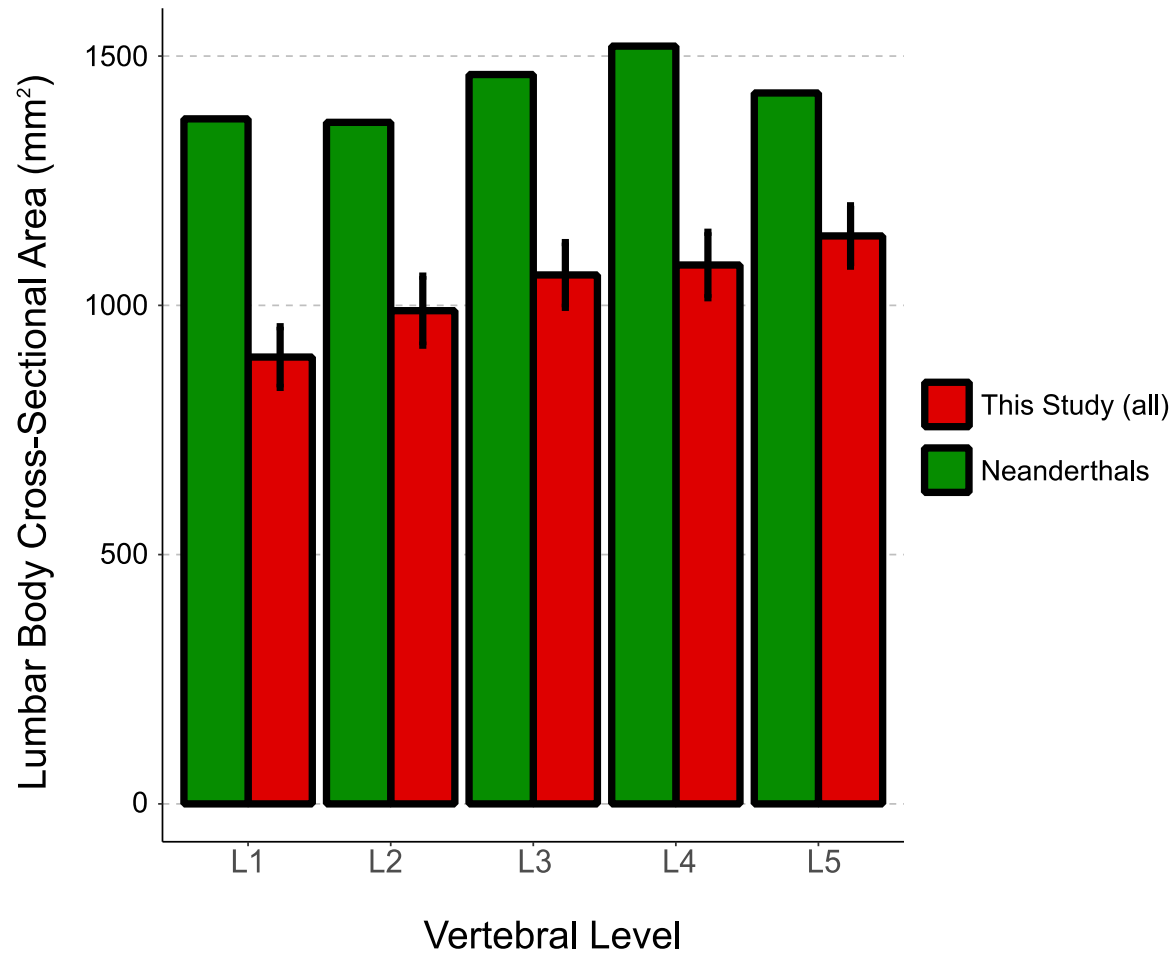


Figure 5.2: Vertebral body surface areas for the lumbar vertebral columns of Neanderthals (green) and cross-sectional areas of modern human lumbar vertebrae (red). Neanderthal values are from the Kebara 2 specimen (measurements reported by Been et al., 2010). Modern human values were measured from MRIs of male and female participants in this study (n = 30). Error bars indicate 95% confidence intervals.

Although the beam model offers one possible explanation for Neanderthal lordosis, the model predicts only 25% of the variation in standing lordosis among study participants. Therefore, most of the variation in lordosis among fossil hominins was likely due to other causes. An alternative explanation is that lordosis variations among hominins reflect adaptations to balance the competing demands of stiffness and stability with lumbar mobility and shock attenuation. As Chapters 3 and 4 showed, this tradeoff may offer a novel framework for understanding the selection pressures acting on the evolution of the hominin lumbar spine. Though having sufficient lordosis was always an important selection

pressure for maintaining postural balance, especially for females during pregnancy (Whitcome et al., 2007), the tradeoff hypothesis predicts that straighter versus more curved spinal postures would have served alternative adaptive functions. On the one hand, Chapter 3 suggests that straighter spines would have functioned better during activities that require spinal stability and resistance to deformations such as carrying heavy loads and for reduced risk of injury due to lower intervertebral shearing forces. On the other hand, Chapter 4 suggests that more curved spines would have functioned better for lumbar shock attenuation during high-impact activities such as running, as well as for activities that require more spinal mobility at the potential cost of increased risk of injury. Thus, static lumbar stiffness and dynamic shock attenuation may represent opposite ends of a spectrum, and variations in posture may trade off to modulate the functional balance of one factor at the expense of the other.

Variability in lordosis observed among modern athletes provides evidence to support this tradeoff hypothesis. Athletes such as sprinters, long distance runners, and soccer players who habitually experience high levels of dynamic, repetitive impacts tend to have greater-than-average lumbar curvature (Uetake and Ohtsuki, 1993; Wodecki et al., 2002). In addition, athletes who rely on high amounts of lumbar flexibility, such as rock climbers, tend to have high degrees of lordosis (Forster et al., 2009). However, athletes such as body builders and swimmers, who do not experience habitually high levels of dynamic shock impacts, tend to have much straighter lumbar spines (Uetake and Ohtsuki, 1993). These individuals often rely on isometric contractions to stabilize their upper bodies, and they tend to perform activities that require spinal stability to support heavy loads (e.g. body builders) or to maintain axial rigidity during dynamic activities (e.g. swimmers),

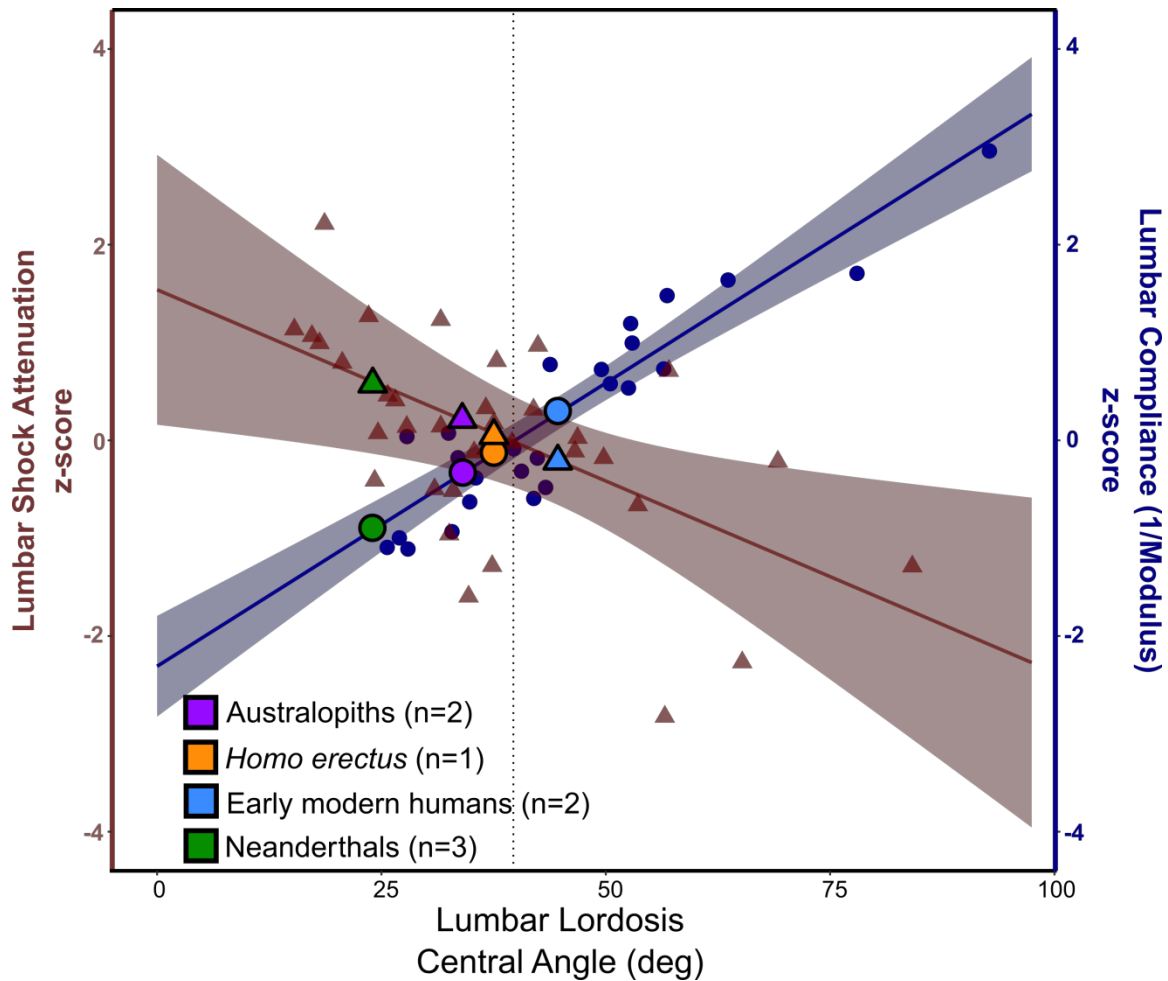


Figure 5.3: Regressions between standing lordosis versus lumbar bending compliance (blue, circles), and standing lordosis versus lumbar shock attenuation during running (red, triangles). To linearize the hyperbolic elastic modulus function, compliance was quantified as the inverse of elastic modulus. Both compliance and shock attenuation were standardized as z-scores for comparison. Shaded regions represent 95% confidence intervals. Shock attenuation increases as z-score values decrease, and compliance increases as z-score values increase. Mean predicted values for hominin taxa are shown for the lumbar compliance (circles) and shock attenuation (triangles) regressions. The point where the regression lines cross was found at 39.7° of lordosis (vertical dotted line).

These data suggest that hominin lordosis variability may reflect adaptations to differential patterns of spinal loading. The tradeoff between static lumbar stiffness and dynamic shock attenuation is apparent from plotting the associations between lordosis, lumbar bending compliance (i.e. the inverse of elastic modulus), and dynamic shock attenuation during running (Figure 5.3). Using predicted lumbar compliance and shock attenuation values based on hominin CA estimates, the position of hominins on the stiffness-attenuation tradeoff permits speculation about the adaptive significance of hominin postural variations.

The stiffness-attenuation hypothesis predicts that lumbar spinal curvature in australopiths may have been shaped by selection for climbing as well as predator avoidance. Though the importance of climbing among early hominins is debated, it is generally believed that the shape of the thorax and features of the upper limb suggest that australopiths were still able to climb effectively (Stern and Susman, 1983). At the same time, it is unlikely that long distance running was a habitual activity among hominins until the emergence of the genus *Homo* (Bramble and Lieberman, 2004), but sprinting for the purposes of predator avoidance likely would have remained an important behavior for australopiths, requiring their spines to attenuate low levels of infrequent dynamic impacts. The tradeoff hypothesis might predict that selection might act to straighten the spine of australopiths in order to increase spinal stiffness and stability, but due to the importance of lordosis during pregnancy (Whitcome et al., 2007), the need for sufficient lumbar compliance for climbing (Forster et al., 2009), and occasional low levels of shock impacts during sprinting, opposing selection pressures would also act to maintain lumbar spinal curvature. Thus, these selection pressures may explain why australopith lordosis (34.2°) was slightly below but nonetheless close to the point at which the relationships between lordosis, shock attenuation, and lumbar stiffness intersect (39.7°) (Figure 5.3).

This tradeoff hypothesis also predicts that *Homo erectus* and later members of the genus *Homo* (e.g. early modern humans) had increased lordosis for endurance running. The evolution of the genus *Homo* is marked by the emergence of hunting and gathering, when endurance running for the purposes of persistence hunting is hypothesized to have led to adaptations throughout the body to cope with dynamic impact forces (Spoor et al., 1994; Bramble and Lieberman, 2004). Thus, it is possible that selection for increased lordosis and climbing-related lumbar compliance in australopiths was superseded by selection for more curved spinal postures and increased lumbar shock attenuation in the genus *Homo*. At the same time, *Homo erectus* and later taxa, as hunter-gatherers, were undoubtedly carrying heavy loads (e.g. infants, tools, and food), which may have led to opposing selection pressures to

maintain sufficiently straight lumbar spines for increased lumbar stiffness and reduced injury risk. These functional constraints may explain why *Homo erectus* (37.5°) and early modern humans (45.1°) had lumbar curves that were near, or slightly above, the optimal value to balance competing functional demands of lumbar stiffness and shock attenuation.

Given the hypothesized selection pressures outlined for other members of the genus *Homo*, the extremely straight lumbar spines of Neanderthals (24.3°) are puzzling. The stiffness-attenuation tradeoff predicts that Neanderthal postures would have been better adapted for spinal stability and stiffness at the expense of reduced lumbar shock attenuation capacity. But the purpose of this postural adaptation is not entirely clear. Some authors have speculated that the straighter lumbar spines of Neanderthals would have been better adapted for carrying loads (Been et al., 2012; Castillo et al., 2017), which is consistent with the findings of this thesis. Though Chapter 3 speculates that the reduced lordosis of Neanderthals could represent an adaptation for carrying external loads or supporting their larger upper bodies (Castillo et al., 2017), the experimental studies in Chapters 3 and 4 provide more convincing evidence that Neanderthal lumbar spines evolved in response to a combination of increased reliance on carrying behaviors combined with reduced selection for endurance running performance.

Several lines of evidence suggest that endurance running performance was reduced in Neanderthals. Endurance running to persistence hunt likely arose (and was most successful) for hunting large animals in hot, arid environments to force prey into hyperthermia (Lieberman et al., 2009). Since Neanderthals generally inhabited very cold climates (many of them forested) compared to early modern humans and other members of the genus *Homo* (Holliday, 1997; Steegman et al., 2002), the effectiveness of persistence hunting would have been greatly reduced for Neanderthals because colder ambient temperatures make it more difficult to drive prey into hyperthermic exhaustion (Carrier, 1984; Bramble and Lieberman, 2004). Therefore, selection for endurance running adaptations, including lumbar shock attenuation capacity, would not have been strong in Neanderthals. Various aspects of

Neanderthal anatomy provide additional evidence of reduced selection for endurance running performance including longer calcaneal tuberosities, increased body mass, relatively short limbs, and smaller, more circular, inferiorly-oriented anterior and posterior semicircular canals (Spoor et al., 2003; Pontzer, 2005, 2007; Steudel-Numbers et al., 2007; Raichlen et al., 2011).

In addition to reduced reliance on endurance running, there is strong evidence from the archaeological record and from foraging models that Neanderthals likely carried heavy loads such as firewood, tools, and offspring. It is also speculated that large game was an important resource often carried by Neanderthals. Faunal data indicate that large herbivores were a crucial part of the Neanderthal diet, and archaeological evidence suggests they were hunting reindeer, bison, aurochs, horses, rhinoceroses, and even mammoths (Stiner, 1994; Gaudzinski and Roebroeks, 2000; Hoffer, 2002; Niven et al., 2012; Demay et al., 2012; Smith, 2015). These data are consistent with stable-isotope analyses from fossil material, which show that the dietary protein consumed by Neanderthals came primarily from large herbivores (Richards and Trinkaus, 2009). Additionally, zooarchaeological evidence suggests that prey were likely butchered at kill sites, and meat was then carried back to camp, requiring Neanderthals (most likely men) to transport heavy burdens over long distances (Niven et al., 2012). Additional evidence suggests that load transport would have been more energetically challenging for Neanderthals. Due to their cold-adapted shorter lower limbs and larger body sizes, daily foraging costs in Neanderthals are estimated to be 188-234 kcal more costly than modern humans (Weaver and Steudel-Numbers, 2005). Given elevated locomotor costs, some foraging models have predicted that Neanderthals would have had a net reduction in foraging returns indicating a reduced foraging radius, and in turn shorter and more frequent foraging trips than modern humans (Verpoorte, 2006). One way Neanderthals could have compensated for reduced caloric returns per foraging trip would have been to increase the mass of food transported during each trip. As Kuhn and Stiner suggest (2006), large game would have provided the highest net return in terms of energetic yield per unit time of any food

resource. By focusing foraging efforts on large game and transporting more massive amounts of the processed carcass over shorter distances back to camp (Macdonald et al., 2009; Verpoorte, 2006), Neanderthals could have maximized foraging returns and compensated for their increased foraging costs.

The hypothesis that Neanderthals were better adapted for load carrying is also supported by fossil evidence of Neanderthal lumbar vertebral wedging. Although intervertebral discs contribute most to lordosis variations in modern adults (Shefi et al., 2013), the sagittal wedge-shaped profile of lumbar vertebral bodies are an important skeletal correlate of lordosis that preserves in the fossil record. Wedging varies along the vertebral column in modern humans, with the difference between the dorsal and ventral height of the vertebral body showing positive values in the upper lumbar (indicating kyphotic wedging) and negative values in the lower lumbar (indicating lordotic wedging) (Figure 5.4). A major factor contributing to the straighter lordosis of Neanderthals is the extreme kyphotic wedging of L1 and L2 (Weber et al., 2008; Been et al., 2010). Using equations by Digiovanni et al. (1989) to calculate vertebral wedging based on ventral and dorsal body heights, modern humans in this study sample are compared to other hominins below (Figure 5.4A). As Figure 5.4 demonstrates, the upper lumbar vertebrae of Neanderthals show much greater kyphotic wedging than any other hominins. Neanderthal L1 wedging angles were 2.3 times greater than the total modern human sample ($p = 0.04$) and 1.9 times greater than modern males ($p = 0.08$), and L2 wedging angles were over 3.5 times greater than the modern sample ($p = 0.02$) and 3.2 times greater than modern males ($p = 0.03$).

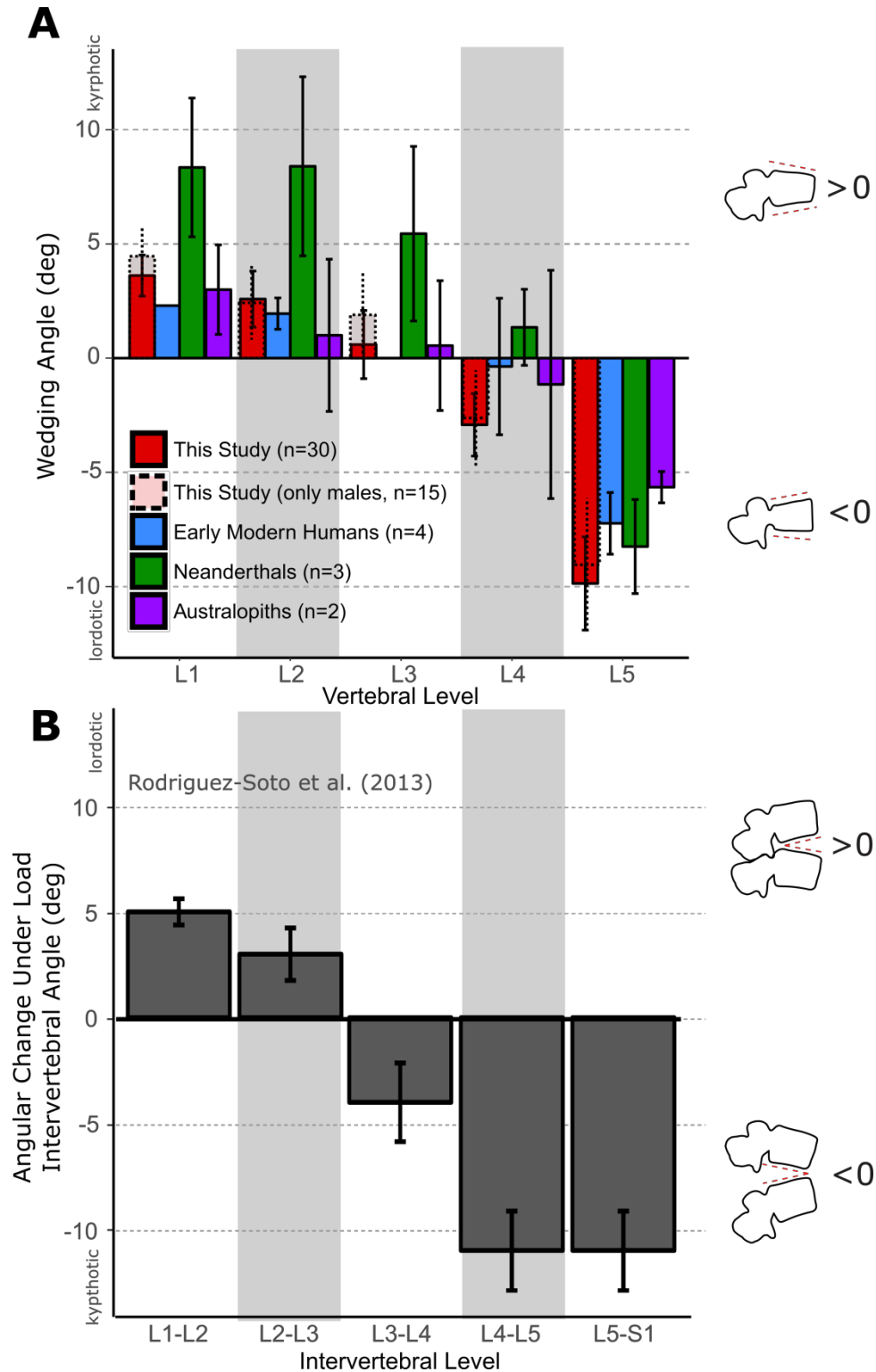


Figure 5.4: (A) Lumbar vertebral body wedging angles calculated following Digiovanni et al. (1989). Positive angles indicate kyrphotic wedging and negative angles indicate lordotic wedging. Data for australopiths are reported in Whitcome et al. (2007), and data for Neanderthals and early modern humans are from Been et al. (2010). (B) Intervertebral angle changes between vertebrae during loading in modern humans carrying 50.8 kg loads for 45 min. Positive angles indicate locally increased lordosis while negative angles indicate locally increased kyrphosis. Data from Rodriguez-Soto et al. (2013). Error bars represent 95% CIs.

The functional significance of Neanderthal vertebral wedging is further illuminated by an experiment that used positional MRIs to study changes in lumbar lordosis and intervertebral angles (the angle between the inferior endplate of one vertebra relative to the superior endplate of the adjacent vertebra) during *in vivo* spinal loading (Rodriguez-Soto et al., 2013). After 45 minutes of loading using a 50.8 kg backpack, study participants showed an interesting pattern of intervertebral rotations in which the upper lumbar spine becomes more curved while the lower lumbar spine becomes straighter under load (Figure 5.4B). In particular, although the average amount of lordosis decreased across study participants, local changes in lordosis showed a pattern where intervertebral angles increased between L1-L2 and L2-L3 (indicating locally increased lordosis), but angles decreased between L4-L5 and L5-S1 (indicating locally increased kyphosis). This pattern of intervertebral change during loading precisely mirrors the pattern of wedging across the hominin lumbar vertebral column, such that intervertebral levels showing the greatest lordotic changes have increased kyphotic vertebral body wedging. But the extreme wedging of the upper lumbar vertebrae of Neanderthals may indicate an adaptation to provide greater resistance to intervertebral rotations in the upper lumbar spine. If Neanderthals show similar patterns of spinal deformation to modern humans, the extreme kyphotic wedged shape of the Neanderthal L1 and L2 would have provided a means to compensate for the increased lordotic intervertebral rotation occurring between the L1-L2 joint, and greater resistance to lordotic changes under load in the upper lumbar spine.

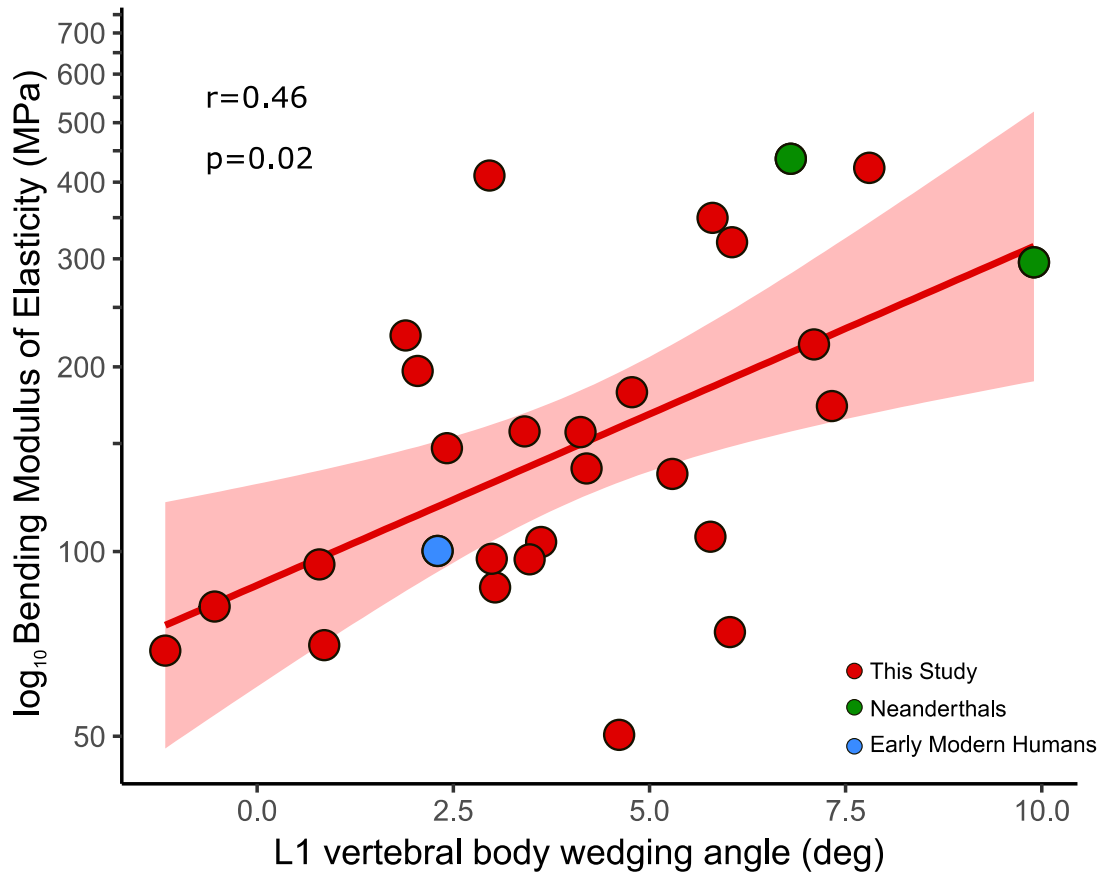


Figure 5.5: The association between L1 vertebral body wedging angles and bending modulus from Chapter 3. For comparison, Neanderthal (Kebara 2 and La Chapelle-Aux-Saints) and early modern human (Cro-Magnon 3) wedging angles and predicted bending moduli (estimated from the modern human regression between modulus and CA) are also shown. The L1 wedging angle explained 21% of the variation in bending modulus among modern humans in this study. These data indicate that the greater kyphotic wedging of L1 might have represented an additional adaptation for carrying loads in Neanderthals. The red shaded area represents the 95% CI of the regression.

To further test this inference, wedging angles as well as predicted bending moduli were examined for Neanderthals, early modern humans, and the participants in this study (Figure 5.5). Though there is not a strong correlation between elastic modulus and the L2 wedging angle ($r = 0.23$, $p = 0.26$), the amount of kyphotic L1 vertebral wedging strongly correlates with elastic modulus and explains 21% of the variation in lumbar bending resistance across modern human study participants ($r = 0.46$, $p = 0.02$). These data provide additional evidence that the pattern of increased “counter wedging” of the upper lumbar spine (especially in L1) of Neanderthals would have provided an anatomical mechanism by

which their spines may have been adapted for spinal stability and greater resistance to lumbar deformations under load.

One final line of fossil evidence that Neanderthals were adapted for carrying comes from their larger lumbar vertebral bodies (Figure 5.2). As discussed above, larger vertebral bodies suggest increased resistance to bending and may be related to the passive consequences of increased upper body dimensions. However, the 25-53% larger lumbar vertebral bodies of Neanderthals would also have provided a greater surface area over which to distribute compression forces during load carrying, thereby reducing the overall stress on the Neanderthal lumbar spine. Collectively, these fossil, archaeological, and experimental data along with foraging models provide multiple and consistent lines of evidence to support the hypothesis that the shape of Neanderthal lumbar spines were the result of relaxed selection for endurance running and increased reliance on load carrying.

Lordosis and the evolution of lower back pain

Results from this thesis also have important implications for the evolutionary origins of human lower back pain (LBP). LBP is a global epidemic and major public health problem today, but its causes remain unclear. It appears that LBP is currently on the rise, affecting an estimated 60-80% of people worldwide at some point in their lives, and it is a leading cause of work loss, disability, and medical visits (Waddell, 2004; Hoy et al., 2014). Though in most cases pain is transitory, remission rates can be as high as 96% and often result in chronic debilitation (Carey et al., 2010). In the U.S. alone, the total economic burden of LBP (including direct and indirect costs) is estimated to be greater than \$118.8 billion per year (Dagenais et al., 2008). Given these staggering numbers, it is astonishing that in 85-95% of cases the specific cause of LBP is unknown, making treatment and prevention exceptionally difficult (Deyo and Weinstein, 2001; Ehrlich, 2003). The epidemiology of LBP is further complicated by the subjectivity of

pain. Pain itself is only a symptom, influenced by a combination of biomechanical, genetic, environmental, and psychosocial factors. Pain may arise from various tissue sources for many different reasons, but even with medical testing and imaging, a precise diagnosis often eludes clinicians (Adams et al, 2006).

Although there are many factors that correlate with LBP—including population history, age, sex, body morphology, overall health, and physical activity—an unknown but likely high percentage of cases may be due to the mechanical consequences of lumbar lordosis. Curvature of the spine generates high levels of shearing forces between adjacent vertebrae and strain at the dorsal pillar of the vertebral column. These shearing forces are resisted by bony features, such as the neural arch and zygapophyses, as well as by soft tissues of the spine including the discs, spinal ligaments, tendons, and muscles (Scholten et al., 1988; Aspden, 1989; Shiraz-Adl et al., 2002; Arjmand and Shirazi-Adl, 2005; Adams et al., 2006; Meakin et al., 2008). Excessive shearing (most often between L5 –S1) can result in painful muscle strain, joint capsule pain, disc herniation, inflammation (spondylitis), degeneration (spondylolysis), and vertebral displacement (spondylolisthesis) (Cyron & Hutton, 1978; Berlemann et al., 1999; Umehara et al., 2000; Kumar et al., 2001; Rajnics et al., 2002; Labelle et al., 2005; Ward and Latimer, 2005; Barrey et al., 2007; Chen et al., 2009).

The relationship between LBP and lordosis has led many researchers to believe that lordosis evolved as an inevitable consequence of bipedal posture at the cost of increased susceptibility to injury and pain due to higher levels of intervertebral shearing forces. However, this hypothesis does not fully explain the rise in modern cases of LBP. An alternative but not mutually exclusive hypothesis is that recent behavioral changes have led to an increase in the prevalence of LBP. Although humans were very physically active for most of our evolutionary history, developed countries have experienced a rise in LBP rates since the Post-Industrial Revolution correlating with an increase in sedentary behaviors like decreased physical activity, prolonged sitting, and changes to the quality and amount of trunk exercise

possibly due to the introduction of novel comforts such as chairs with back supports and soft, plush mattresses (Lieberman, 2013; Castillo and Lieberman, 2015). Indeed, differences in the magnitude and frequency of spinal loading may explain why active farmers from low-income countries reportedly have 2-4 times lower rates of LBP than sedentary, urban people from high-income countries (Volinn, 1997). Thus, this alternative mismatch hypothesis posits that some cases of LBP are due to an increase in sedentary behaviors that have led to weakened trunk muscles, spinal instability, and increased risk of pain (Castillo and Lieberman, 2015).

As potential support for this hypothesis, Adams et al. (2006) speculated that physical activity and fitness may vary with spinal injury risk in a U-shaped curve. At one extreme, low levels of physical activity and weak spinal tissues (common in very physically inactive populations) may lead to higher risk of injury due to muscle atrophy and loss of mechanical stability. At the other extreme, very high levels of spinal loading may increase the risk of injury due to tissue degeneration and fatigue—a pattern explaining the correlation between LBP and high amounts of occupational loading. Between these extremes, moderate physical activity and spinal loading may lead to stronger back tissues, more stability, and minimal risk of injury. Moderate spinal loading is probably typical of hunter-gatherers and thus provides a normative framework for understanding patterns of spinal loading through human evolution. Since most people in Western populations are relatively inactive, however, this model explains that the majority of LBP cases today result from muscle weakness and low spinal loading, supporting the mismatch hypothesis presented here.

The mismatch hypothesis is further supported by an emerging body of evidence suggesting that most cases of LBP today are the result of muscular dysfunction. Muscles play an essential but poorly understood role in LBP onset. Back muscle strain or sprain could drive up to 70% of LBP cases (Deyo and Weinstein, 2001). Evidence suggests that people with LBP tend to develop atrophied deep trunk muscles (especially the multifidus), more fatigue-prone back muscle fibers, and different patterns of trunk

muscle activation compared with healthy controls (Mannion et al., 1997; Kader et al., 2000; Nelson-Wong et al., 2008). Physical therapy including strengthen trunk muscles thus remains one of the most effective, commonly prescribed, and conservative treatments of nonspecific LBP today (Slade and Keating, 2006; Gatti et al., 2011). But there is only limited understanding of how and why exercise is effective.

Findings reported in this thesis may shed light on the role of muscles in LBP onset. The interaction between relative trunk strength and lumbar flexibility reported in Chapter 2 suggests that muscle strength has a strong influence on lumbar lordosis and spinal range of motion, two features known to affect LBP (Adams et al., 2006). If the factors driving variations in lordosis between individuals also function to adjust lumbar posture within an individual, then Chapter 2 may suggest that exercises to strengthen the abdominal or back muscles can help achieve less injury-prone lumbar postures. Additionally, the strength-flexibility tradeoff predicts that abdominal strengthening, which is particularly associated with straighter lumbar spines, may be effective for reducing intervertebral shearing. Although it is not clear whether “flatback” syndrome is a reactive symptom or a cause of LBP, straighter lumbar spines are often observed among individuals with LBP (Adams et al., 2006), and this thesis may provide a mechanistic explanation behind how and why individuals develop straighter lumbar spines during cases of LBP in order to reduce intervertebral shearing forces and achieve other biomechanical benefits of straighter lumbar postures (Adams and Hutton, 1985). An additional clinical implication of the strength-flexibility model is that trunk exercises to modulate lumbar posture would be ineffective among individuals with immobile lumbar spines, such as those having undergone lumbar spinal fusion. Although the preservation of spinal mobility is often a goal following surgery, the interaction between trunk strength and flexibility may explain why intervention studies to increase spinal stabilization while simultaneously maintaining mobility often result in unsuccessful outcomes (Shymon et al., 2014).

Likewise, results from Chapters 3 and 4 indicate that differences in posture have functional consequences. The association between lumbar posture, stability, and strength have important implications for potential causes of LBP due to variations in core muscle strength. If straighter spines are more resistant to bending deformations, and straighter lumbar spines are also associated with increased abdominal strength, then these results may explain the correlation often found between core abdominal weakness and LBP prevalence (e.g. Ferreira et al., 2004). In addition, the dynamic experiments in this thesis show that curved spines are associated with increased dynamic mobility and shock attenuation (Chapter 4). In addition to greater shearing forces, curved spinal postures are associated with higher dynamic spinal deformations that could lead to increased tissue strain, which may be another potential source of LBP.

Conclusion and future directions

This thesis provides a novel framework for interpreting lordosis in extant and extinct hominins and for understanding the potential origins of lower back pain. From an evolutionary perspective, the results presented here provide support for the hypothesis that some of the variation in hominin lumbar lordosis is explained by differences in body size and shape, with expanded dimensions of the Neanderthal thorax indicating increased bending moments to straighten the lumbar spine. This conclusion is further supported by fossil evidence that Neanderthals had larger lumbar vertebral bodies compared to modern humans. But the passive effects of trunk geometry explain only a small proportion of the overall lordosis variability in modern humans. Therefore, another hypothesis is that postural differences among hominins reflect adaptations for different patterns of spinal loading. The experimental data presented in this thesis suggest that hominin lumbar spines were shaped by a tradeoff between static lumbar bending resistance and dynamic shock attenuation capacity. This tradeoff suggests that straighter Neanderthal lumbar spines were better adapted for spinal stability and

bending resistance. Using evidence from paleontological, archaeological, and modern biomechanical data, Neanderthals are hypothesized to show signals of reduced selection for endurance running performance as well as increased selection for carrying heavy loads. This hypothesis is also supported by the pattern of lumbar vertebral body wedging in Neanderthals, which show extreme kyphosis in the upper lumbar vertebrae as well as overall increased lumbar vertebral body size, which would have provided increased surface area over which to distribute forces. Paired with previous experimental data of intervertebral deformations during loading and the correlation between L1 body wedging and lumbar bending modulus in this study, it is likely that Neanderthal lower backs were specially adapted to provide greater resistance to bending deformations particularly in the upper region of lumbar vertebral column, suggesting selection for carrying behaviors. Though more data are needed, evidence strongly supports the hypothesis that lumbar lordosis variability through hominin evolution was adaptive.

The evolution of hominin lumbar lordosis also may have led to biomechanical vulnerabilities that increase the risk of spinal injury and pathology due to higher levels of intervertebral shearing forces. Variations in lordosis within modern humans have functional consequences explained by several factors including trunk mobility and muscle strength, which are furthermore influenced by physical activity and exercise. Thus, it is possible that the rise in lower back pain in part may be due to increased sedentary behaviors and muscle weakness, leading to spinal instability and pain.

A future direction of this project should focus on the role of muscles. Muscle activation is a critical factor that affects standing postural stability as well as static and dynamic loading behavior. Although we measured overall muscle size in Chapter 2, this thesis did not study active muscle recruitment during any experiments. Muscle activity almost certainly affects lumbar bending modulus and shock attenuation, and future studies would greatly benefit from conducting these experiments while incorporating electromyographic measures of trunk hypaxial and epaxial muscle co-activation.

These data would also provide critical information related to LBP. Several questions remain about the role of muscles in LBP onset, particularly regarding the direction of causality. For example, do people with weaker, more fatigable muscles show changes in spinal posture and instability over time, which then lead to injury and back pain? Or do people first develop LBP for other reasons, which then lead to compensatory spinal postures, reduced lumbar mobility, muscle atrophy, and changes in muscle fiber composition due to disuse and pain avoidance? Given the abundance of evidence for the importance of spinal stability in LBP onset, the role of muscle recruitment is a crucial factor that remains to be tested. Long-term prospective, randomized-controlled trials would be the best way to study which factors related to muscular dysfunction (e.g. more fatigue-prone back muscle fibers, patterns of trunk muscle activation, overall muscle weakness) are causally associated with LBP.

References

- Adams MA, Hutton WC. 1985. The effect of posture on the lumbar spine. *J Bone Joint Surg* 67(4): 625-9.
- Adams MA, Bogduk N, Burton AK, Dolan P. 2012. *The Biomechanics of Back Pain*. Edinburgh: Churchill Livingstone.
- Been E, Gómez-Olivencia A, Kramer PA. 2012. Lumbar lordosis of extinct hominins. *Am J Phys Anthropol* 147: 64–77.
- Bokduk N, Macintosh JE, Pearcy MJ. A universal model of the lumbar back muscles in the upright position. *Spine* 1992; 17:897-913.
- Bramble DM, Lieberman DE. 2004. Endurance running and the evolution of Homo. *Nature* 432(7015): 345-52.
- Carey TS, Garrett JM, Jackman AM. 2000. Beyond the good prognosis. Examination of an inception cohort of patients with chronic low back pain. *Spine* 25(1): 115–20.
- Carrier DR. 1984. The energetic paradox of human running and hominid evolution [and comments and reply]. *Curr Anthropol* 25(4): 483-95.
- Castillo ER, Lieberman DE. 2015. Lower back pain. *Evol Med Public Health* 1: 2-3.
- Cyron BM, HuttonWC. 1978. The fatigue strength of the lumbar neural arch in spondylolysis. *J Bone J Surg* 60(2): 234-238.
- Dagenais S, Caro J, HaldemanS. 2008. A systematic review of low back pain cost of illness studies in the United States and internationally. *Spine J* 8(1): 8-20.
- Demay L, Péan S, Patou-Mathis M. 2012. Mammoths used as food and building resources by Neanderthals: Zooarchaeological study applied to layer 4, Molodova I (Ukraine). *Quat Int* 276: 212-26.
- Deyo RA, Weinstein JN. 2001. Low back pain. *N Engl J Med* 344(5): 363-70.
- Digiovanni B, Scoles P, Latimer B. 1989. Anterior extension of the thoracic vertebral bodies in Scheuermann's kyphosis: an anatomic study. *Spine* 14: 712-6.
- Ehrlich GE. 2003. Back pain. *J Rheumatol* 67: 26-31.
- Ferreira PH, Ferreira ML, Hodges PW. 2004. Changes in recruitment of the abdominal muscles in people with low back pain: ultrasound measurement of muscle activity. *Spine* 29(22): 2560-6.
- Forster R, Penka G, Bosl T, Schoffl VR. 2009. Climber's back-form and mobility of the thoracolumbar spine leading to postural adaptations in male high ability rock climbers. *Int J Sports Med* 30: 53–9.
- Gatti R1, Faccendini S, Tettamanti A, Barbero M, Balestri A, Calori G. 2011. Efficacy of trunk balance exercises for individuals with chronic low back pain: a randomized clinical trial. *J Orthop Sports Phys Ther* 41(8): 542-52.

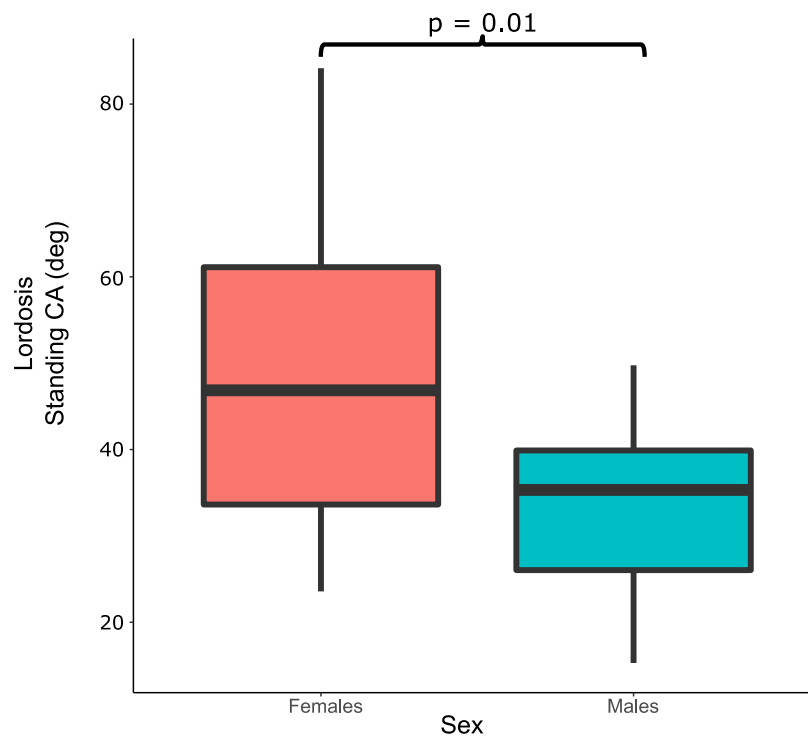
- Gatti R, Faccendini S, Tettamanti A, Barbero M, Balestri A, Calori G. 2011. Efficacy of trunk balance exercises for individuals with chronic low back pain: a randomized clinical trial. *J Orthop Sports Phys Ther* 41(8): 542-5.
- Gaudzinski S, Roebroeks W. 2000. Adults only. Reindeer hunting at the middle palaeolithic site Salzgitter Lebenstedt, northern Germany. *J Hum Evol* 38(4): 497-521.
- Holliday TW. 1997. Postcranial evidence of cold adaptation in European Neandertals. *Am J Phys Anthropol* 104(2): 245-58.
- Hoy D, March L, Brooks P, Blyth F, Woolf A, Bain C, Williams G, Smith E, Vos T, Barendregt J, Murray C. 2014. The global burden of low back pain: estimates from the Global Burden of Disease 2010 study. *Ann Rheum Dis* 73(6): 968-74.
- Kader D, Wardlaw D, Smith F. 2000. Correlation between the MRI changes in the lumbar multifidus muscles and leg pain. *Clin Radiol* 55: 145-9.
- Kuhn S, Stiner M, BarOz G, WeinsteinEvron M, BocquetAppel J, Hovers E, MacDonald K, Roebroeks W, Martnez K, Shea J, Soffer O. 2006. What's a mother to do? The division of labor among Neandertals and modern humans in Eurasia. *Curr Anthropol* 47(6): 953-81.
- Lieberman DE. 2013. *The story of the human body: Evolution, health, and disease*. New York: Pantheon.
- Lieberman DE, Bramble DM, Raichlen DA, Shea JJ. 2009. Brains, brawn, and the evolution of human endurance running capabilities. In: Grine, FE, Fleagle JG, Leakey RE (eds.). *The First Humans: Origins and Early Evolution of the Genus Homo*. New York: Springer. pp. 77-92.
- Macdonald K, Roebroeks W, Verpoorte A. 2009. An energetics perspective on the Neandertal record. In: *The evolution of hominin diets Netherlands*. Springer. pp. 211-20.
- Mannion, A.F., Dumas, G.A., Cooper, R.G., Espinosa, F.J., Faris, M.W. and Stevenson, J.M., 1997. Muscle fibre size and type distribution in thoracic and lumbar regions of erector spinae in healthy subjects without low back pain: normal values and sex differences. *J Anat* 190(4): 505-13.
- Nelson-Wong E, Gregory DE, Winter DA, Callaghan JP. 2008. Gluteus medius muscle activation patterns as a predictor of low back pain during standing. *Clin Biomech* 23(5): 545-553.
- Niven L, Steele TE, Rendu W, Mallye JB, McPherron SP, Soressi M, Jaubert J, Hublin JJ. 2012. Neandertal mobility and large-game hunting: the exploitation of reindeer during the Quina Mousterian at Chez-Pinaud Jonzac (Charente-Maritime, France). *J Hum Evol* 63(4): 624-35.
- Pontzer H. 2005. A new model predicting locomotor cost from limb length via force production. *J Exp Biol* 208(8): 1513-24.
- Pontzer H. 2007. Predicting the energy cost of terrestrial locomotion: a test of the LiMb model in humans and quadrupeds. *J Exp Biol* 210(3): 484-94.
- Potvin JR, Norma RW, McGill SM. 1991. Reduction in anterior shear forces on the L4/L5 disc by the lumbar musculature. *Clin Biomech* 6: 88-96.

- Raichlen DA, Armstrong H, Lieberman DE. 2011. Calcaneus length determines running economy: implications for endurance running performance in modern humans and Neandertals. *J Hum Evol* 60(3): 299-308.
- Richards MP, Trinkaus E. 2009. Isotopic evidence for the diets of European Neanderthals and early modern humans. *Proc Natl Acad Sci USA* 106(38): 16034-9.
- Rodriguez-Soto AE, Jaworski R, Jensen A, Niederberger B, Hargens AR, Frank LR, Kelly KR, Ward SR. 2013. Effect of load carriage on lumbar spine kinematics. *Spine* 38(13): E783-91.
- Schultz AH. 1961. Vertebral column and thorax. *Primatologia* 4, 1–66.
- Schwarzer AC, Wang SC, Bogduk N, McNaught PJ, Laurent R. 1995. Prevalence of clinical features of lumbar zygapophyseal joint pain: a study in an Australian population with chronic low back pain. *Ann Rheum Dis* 54: 100-6.
- Shefi S, Soudack M, Konen E, Been E. 2013. Development of the lumbar lordotic curvature in children from age 2 to 20 years. *Spine* 38(10): E602-8.
- Slade SC, Keating JL. 2006. Trunk-strengthening exercises for chronic low back pain: a systematic review. *J Manipulative Physiol Ther* 29(2):163-73.
- Smith GM. 2015. Neanderthal megafaunal exploitation in Western Europe and its dietary implications: a contextual reassessment of La Cotte de St Brelade (Jersey). *J Hum Evol* 78: 181-201.
- Spoor, F., Wood, B., Zonneveld, F., 1994. Implications of early hominid labyrinth morphology for the evolution of human bipedal locomotion. *Nature* 369: 645-8.
- Stegmann AT, Cerny FJ, Holliday TW. 2002. Neanderthal cold adaptation: physiological and energetic factors. *Am J Hum Bio* 14(5): 566-83.
- Stern JT, Susman RL. 1983. The locomotor anatomy of *Australopithecus afarensis*. *Am J Phys Anthropol* 60: 279–317.
- Steudel-Numbers KL, Weaver TD, Wall-Scheffler CM. 2007. The evolution of human running: effects of changes in lower-limb length on locomotor economy. *J Hum Evol* 53(2): 191-6.
- Stiner MC. 1994. Honor among thieves: A zooarchaeological study of Neanderthal ecology. Princeton: Princeton University Press.
- Thomas E, Silman AJ, Croft PR, Papageorgiou AC, Jayson MI, Macfarlane GJ. 1999. Predicting who develops chronic low back pain in primary care: a prospective study. *Bos Med J* 318(7199): 1662-7.
- Verpoorte A. 2006 . Neanderthal energetics and spatial behaviour. *Before Farming* 3: 1-6.
- Volinn E. 1997. The epidemiology of low back pain in the rest of the world. *Spine* 22: 1747-54.
- Wadell G. 2004. *The Back Pain Revolution*. Edinburgh: Churchill Livingstone.
- Weber J, Pusch CM. 2008. The lumbar spines of Neanderthals shows natural kyphosis. *Eur Spine J* 17(S2): S327-30.

Appendix 1 – Supplementary Data

Sex differences in lumbar lordosis

Lumbar lordosis is known to covary with sex and is therefore an important factor that could affect the results presented here. The main outcome variable used to measure lordosis throughout this thesis was the Standing Central Angle (CA). Among the sample of participants measured in this study (n=30), Standing CA was 46% greater in males compared to females ($p = 0.01$) (Appendix Figure 1.1).



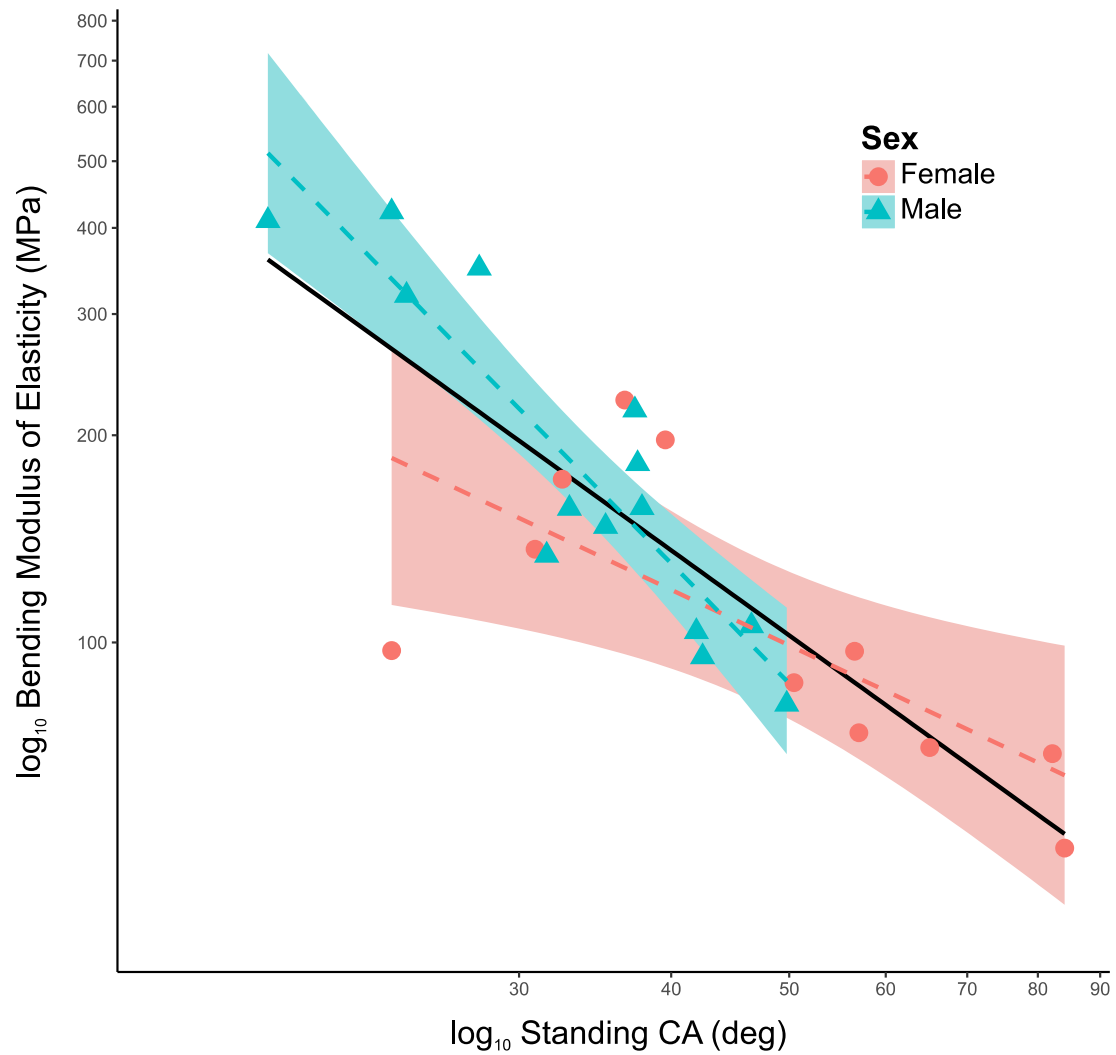
Appendix Figure 1.1: Comparison of Standing Central Angle (CA) between male and female participants in this study (n=30). Females (pink) had 46% greater Standing CA than males (blue) on average.

However, when including sex as a covariate, the main results reported from Chapters 2 through 4 were unchanged. Sex had no effect on the strength-flexibility tradeoff model in Chapter 2 ($p = 0.58$), no effect on the relationship between Standing CA and the bending modulus of elasticity in Chapter 3 ($p = 0.38$)

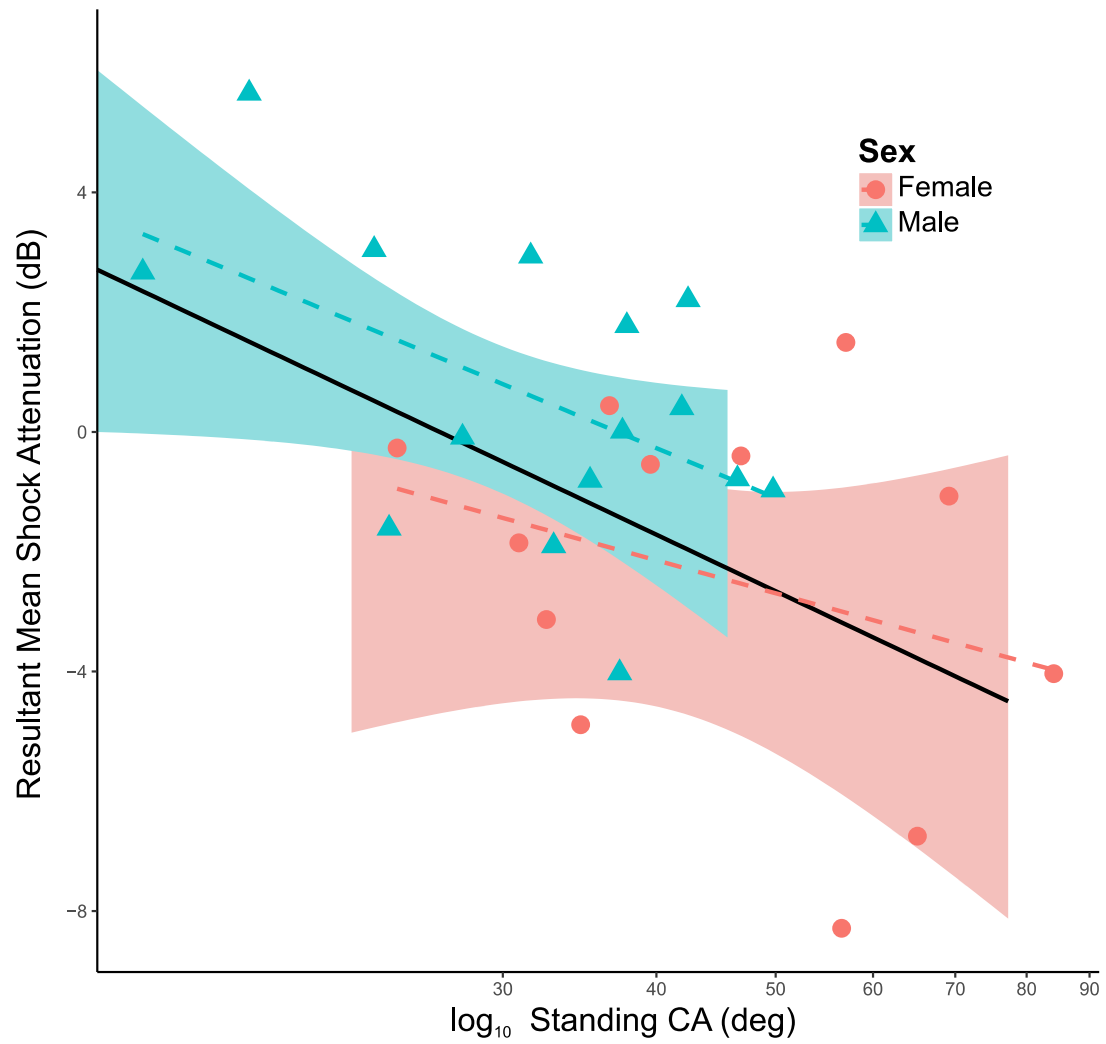
(Appendix Table 1.1; Appendix Figure 1.2), and no effect on the relationship between Standing CA and resultant mean shock attenuation during running in Chapter 4 ($p = 0.09$) (Appendix Table 1.1; Appendix Figure 1.3).

Appendix Table 1.1: The main findings from Chapters 2-4 controlling for sex as an additional covariate.

Chapter 2: Standing CA versus the strength-flexibility model controlling for sex as a covariate			
	<u>β</u>	<u>S.E.</u>	<u>p-value</u>
(Intercept)	0.03	0.11	0.81
$\log_{10}$ (Relative Trunk Strength)	-0.40	0.12	0.003
$\log_{10}$ (Range of Motion)	0.75	0.16	0.00007
Sex	-0.08	0.15	0.58
$\log_{10}$ (Relative Trunk Strength) : $\log_{10}$ (Range of Motion)	-0.25	0.09	0.01
Multiple R-squared:	0.66		
Chapter 3: Standing CA versus bending modulus of elasticity controlling for sex as a covariate			
	<u>Coef.</u>	<u>S.E.</u>	<u>p-value</u>
(Intercept)	4.02	0.34	<0.000001
$\log_{10}$ (Standing CA)	-1.19	0.20	0.000006
Sex	0.06	0.07	0.38
Multiple R-squared:	0.70		
Chapter 4: Standing CA versus running resultant shock attenuation controlling for sex as a covariate			
	<u>Coef.</u>	<u>S.E.</u>	<u>p-value</u>
(Intercept)	9.36	5.49	0.10
$\log_{10}$ (Standing CA)	-7.13	3.29	0.04
Sex	1.94	1.10	0.09
Multiple R-squared:	0.37		



Appendix Figure 1.2: Association between Standing Central Angle (CA) and the lumbar bending modulus of elasticity from Chapter 3. The regressions for males (blue) and females (pink) are shown with 95% confidence intervals shaded. The overall model regression is also shown (black solid line). Although females had larger CAs than males, there was no difference in the regression lines between sexes (Appendix Table 1.1).



Appendix Figure 1.3: Association between Standing Central Angle (CA) and resultant mean shock attenuation from Chapter 4. The regressions for males (blue) and females (pink) are shown with 95% confidence intervals shaded. The overall model regression is also shown (black solid line). Although females had larger CAs than males, there was no difference in the regression lines between sexes (Appendix Table 1.1).